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CONTENTS

	Page
Thromboangiitis Obliterans of Patients with Diabetes. BAYARD T. HORTON, and FRANK N. ALLAN	799
Human Constitution. A Study of the Correlations between Physical Aspects of the Body and Susceptibility to Certain Diseases. WALTER FREEMAN	805
Paroxysmal Ventricular Tachycardia: An Etiological Study with Special Reference to the Type. CLAYTON J. LUNDY and LAWRENCE L. McLELLAN	812
Fatal Tularemia. Review of Autopsied Cases with Report of a Fatal Case. LEWIS P. GUNDRY and C. GARDNER WARNER	837
Fragilitas Ossium in Five Generations. HENRY JOACHIM and MILTON G. WASCH	853
A Justification of the Diagnosis of Chronic Nervous Exhaustion. JOHN W. MACY and EDGAR V. ALLEN	861
Irradiation Treatment of Hyperthyroidism. GEORGE E. PFAHLER	868
The Cause of Death of Patients with Organic Heart Disease Subjected to Surgical Operation. W. K. PURKS	885
Myeloid Insufficiency. R. GOTTLIEB	895
The Importance of Bronchoscopy in Bronchiectasis. HORACE P. MARVIN	903
Editorial	913
Reviews	916
College News Notes	919
Obituaries	921
Abstract of Minutes of the Board of Regents	923

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ANNALS OF
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THROMBOANGIITIS OBLITERANS OF PATIENTS WITH DIABETES*

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GANGRENE of the lower extremities in cases of diabetes is due primarily to impairment of circulation and secondarily to the effects of the diabetes on the resistance of the skin to infection. In cases of long standing, without adequate treatment, arteriosclerosis is common. It develops early in life and to a more advanced degree than in nondiabetic patients. In some cases, however, the occlusive process is of a different type. The present report, with the exception of a case reported by Adams in 1930, includes all cases of thromboangiitis obliterans complicated with diabetes observed at The Mayo Clinic. The small number indicates the rarity of the association of these diseases.

CASE I

A Russian Jew, aged 42 years, registered at The Mayo Clinic May 1, 1930. His chief complaints at the time of admission were diabetes and pain in the feet, which had been present, respectively, for five months and 18 years. Eighteen years prior to his admission, he first had noticed intermittent claudication in the calf of the left leg while drilling with the Marine Corps. The pain came on only while he was walking, and relief was obtained with rest. This symptom was slowly progressive for many years. Seven years before his admission, the pain of claudication had begun to develop after he had walked briskly two or three blocks, and was present in the muscles of both calves. Five months before his admission, a fissure had developed between the third and fourth toes of the left foot; pain while at rest had developed, and this was rather severe at the time of his examination. He had not attempted to walk during these five months, and had been in bed practically constantly. About 0.5 grain (0.032 gm.) of morphine had been required at night to relieve the pain. Fourteen years before his examination, sugar had been discovered in the urine. He had tried repeatedly to obtain life insurance following that discovery, but had been rejected by the examining physician at each attempt. He had never consulted a physician regarding the diabetes

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From the Division of Medicine, The Mayo Clinic.

until the development of the ulcer on the left foot. Polydipsia and polyuria had never been important features in his disorder. He had been taking 10 units of insulin three times a day for three weeks before his admission. He had smoked 15 cigarettes daily for many years, and had used alcohol in relatively large quantities for 12 years. There was no history of superficial phlebitis.

The man was well developed, 5 feet 6 inches (167 cm.) in height and weighed 150 pounds (68 kg.). He had lost 30 pounds (13.6 kg.) since the development of pain in the left foot while at rest. He appeared to be exhausted. The blood pressure was 90 mm. of mercury systolic, and 62 diastolic. Examination of urine gave negative results, except for the presence of albumin, graded 1. The blood counts were normal and the Wassermann test of the blood was negative. The tonsils were infected, and tonsillectomy was performed. The value for blood sugar was 0.130 gm. in each 100 c.c. and repeated determinations of blood sugar during the patient's 27 days' stay in the hospital gave values within normal limits or only slightly above normal. The highest value for blood sugar following hospitalization was 0.170 and 0.160 gm. in each 100 c.c. The fissure, previously mentioned, was 1.5 cm. long. Pulsations of the palpable arteries of the hands were normal, except those of the right ulnar artery, which were reduced 50 per cent. Both femoral and both popliteal arteries pulsated normally. Both posterior tibial and dorsalis pedis arteries were occluded. Pallor of the feet followed elevation of them; that of the right foot was graded 3 and that of the left foot, 2. Rubor, graded 2, was present when the feet were dependent. Roentgenograms of the legs gave evidence of mild arteriosclerosis. The vasomotor index of the left foot ranged from 1.3 to 2 and that of the right foot from 1.8 to 2.4 (low values).

The diet prescribed consisted of 73 gm. of carbohydrate, 53 gm. of protein, and 176 gm. of fat. The patient continued to take 10 units of insulin three times daily. During his stay of 27 days in the hospital he received seven intravenous injections of typhoid vaccine, and at the time of his dismissal the ulcer had entirely healed. He was dismissed with directions to take daily, 77 gm. of carbohydrate, 50 gm. of protein, 144 gm. of fat, and 40 units of insulin. The patient died two years later of morphine addiction combined with an acute psychosis.

The diagnosis of thromboangiitis obliterans was valid because the patient was young (aged 24 years) at the time of onset of symptoms, because the circulation in the feet was diminished, and because pulsations of the right ulnar artery were impaired. The presence of slight grades of calcification of the arteries of the legs indicated a mixed occlusive process of the vessels. The arteriosclerotic changes were probably accelerated by the presence of diabetes.

Case II

A man, aged 47 years, of English-American stock, was admitted to hospital in August 1930, because of pains in the left leg and foot, and a gangrenous ulcer on the left fifth toe. He had begun to have pains of claudication in the left foot in April 1930; later he had pains when at rest, and the trouble had become worse, so that he had become completely disabled. A year before admission a corn had been removed by a chiropodist, leaving an ulcer on the toe which had failed to heal. A diagnosis of diabetes mellitus had been made in September 1929, because of the discovery of glycosuria. The intake of carbohydrate had been restricted for a short time, but for several months before the patient's admission to the clinic he had been eating ordinary meals, including some sugar and pastry. The urine had been free from sugar and the concentration of sugar in the blood, normal. The patient was given the ordinary hospital diet for three weeks. In this period, a trace of sugar was found in the urine

on two occasions, and the value for blood sugar was 130 mg. for each 100 c.c., also on two occasions. In order to verify the diagnosis of diabetes a sugar tolerance test was made. The response was typical of mild diabetes. The concentration of sugar in the blood rose from 0.120 to 0.240 gm. in each 100 c.c. in half an hour, and to 0.310 in two hours. From this time on restriction of the carbohydrate of the diet was advised. The patient had been smoking 40 cigarettes a day for 10 years. He had had symptoms suggesting renal colic, but his previous health had otherwise been satisfactory.

Examination at the clinic disclosed absence of pulsations of the arteries of the left foot. Pulsations of the usually palpable arteries of the other extremities appeared normal. There was abnormal pallor of the foot when elevated. Roentgenologic examination of the feet and legs did not give evidence of calcification of the vessels. The other examinations gave negative results.

Efforts were made to improve the circulation of the foot by induction of fever and other measures. The patient went home, but returned in six weeks with gangrene of the left foot, suffering intense pain. A Gritti-Stokes amputation of the leg was performed September 29, 1930. The following day, sugar appeared in the urine, and treatment with insulin was instituted. The amount of insulin had to be increased daily until the ninth day, when a dose of 80 units was reached. The amount of insulin given daily had gradually been reduced to 15 units on the twenty-fourth day after operation. The stump failed to heal, and it was necessary to amputate again, through the thigh, November 3, 1930. Aggravation of the diabetes was again encountered, and larger doses of insulin were required for 10 days, but the dose given was not so large as after the first operation. The use of insulin was discontinued on the eighteenth day after the second amputation. Satisfactory healing of the wound finally occurred. The urine remained free from sugar, and the concentration of sugar in the blood remained normal on a diet of 135 gm. of carbohydrate. Only sugar and sweet desserts were omitted from the diet. The diabetes had again become latent.

The patient was seen again July 19, 1932. At this time he was in good health. The stump of the left thigh was in good condition. The right foot seemed normal except for slight diminution in the pulsations in the dorsalis pedis and posterior tibial arteries. The color of the foot was normal. Dietary regulation had maintained control of the diabetes. The patient was advised to continue precautions to protect his foot from injury. He was also advised not to smoke.

Concerning case 2, attention should be directed to the fact that before operation diabetes was practically latent, although the patient had been on an ordinary diet for a long period and the urine was free from sugar, except for a trace on rare occasions. The value for blood sugar was just on the borderline of the normal range. Under these conditions the existence of diabetes might be doubted, or it might be thought that diabetes, if present, was so mild as to be of no significance. The diagnosis of diabetes was confirmed by the glucose tolerance test, and the events following operation showed that the condition was by no means benign. Under ordinary conditions, the diabetes was so mild it could be detected only by a glucose tolerance test; following the surgical operation it was changed to diabetes of severest grade. The amount of insulin needed was as high as that required in the most severe cases.

The diagnosis of thromboangiitis obliterans was based on the age of the patient, absence of calcification of the arteries of the affected extremity, and a history of excessive use of cigarettes. This diagnosis was confirmed by a

study of the anterior and posterior tibial arteries of the amputated extremity. Numerous segments were occluded with old and recent thrombi. The recent lesions were characterized by a chronic inflammatory process. Giant cells were present in small numbers in some of the small, occluded vessels. Calcification of the walls of the vessels was not encountered in the sections studied.

Case III

A Russian Jew, aged 38 years, registered at the clinic July 9, 1926, because of the following conditions: diabetes mellitus which had been present in mild form for seven years; twitching and coarse jerking movements involving various groups of muscles, which had been present for one year, and intermittent pain of claudication of the left leg, which had developed three months before admission. At the time the diabetes was discovered, the man's intake of carbohydrate had been restricted, and the urine at once had become free of sugar and had remained so for two years. He then had ceased to follow the prescribed diet, and glycosuria again had developed. He had then been given 18 units of insulin daily, and a diet of fats 165 gm., protein 65 gm., and carbohydrates 150 gm., which he had continued to follow. The jerking and twitching of the various groups of muscles, especially of the muscles of the legs and arms, usually developed at night, frequently after a hard day's work. This had not been progressive. The pain of intermittent claudication in the muscles of the left calf had become progressively worse. It was definitely associated with exercise. It came on after walking a half block to a block; with further walking, the pain became worse, spread up the leg, and the man became lame. Rest gave relief of symptoms. There was no history of phlebitis. The patient smoked about 18 cigarettes daily.

General examination gave essentially negative results, except that the left foot was distinctly colder than the right. Pulsations, however, were present in both dorsalis pedis and posterior tibial arteries, and no definite rubor or blanching of the feet was observed. Unfortunately, no record was made of the pulsations in the upper extremities at that time, for we were not as familiar with thromboangiitis obliterans as we are at present. The blood pressure was 120 mm. of mercury systolic, and 70 diastolic. The urine at the time of the patient's admission was negative, the blood counts were normal and the Wassermann test of the blood was negative. The value for blood sugar was 0.110 gm. in each 100 c.c. Roentgenograms of the thorax, teeth and nasal accessory sinuses gave negative results. Roentgenograms of the extremities were not made. Neurologic examination gave objectively negative results. Foci of infection were not present. The sugar tolerance test gave evidence of mild diabetes mellitus.

The patient was sent home, with directions to take 72 gm. of carbohydrate, 50 gm. of protein, and 188 gm. of fat daily, without insulin. We have been unable to obtain response to follow-up letters from this patient.

A tentative diagnosis of early thromboangiitis obliterans and diabetes mellitus was made. The history of intermittent claudication alone suggested an occlusive vascular process in the left leg, which apparently involved largely the muscular branches. The age and race of the patient aided in the diagnosis.

Case IV

A Russian Jew, aged 42 years, first came to the clinic October 24, 1930, because of pain of three months' duration, in the first and second right toes, when at rest. An

ulcer had developed on the first toe at that time. Superficial phlebitis of the left leg first had been observed three years before the patient's admission to the clinic, and a similar condition of the right leg, one year before his admission to the clinic. Intermittent pain of claudication, involving the arch of the right foot, had been observed for one year. This had become progressively worse, and the man had been able to walk only a half block without pain at the time the ulcer on the right great toe developed. He had smoked 20 cigarettes daily for 20 years.

Physical examination was negative, except with respect to the lower extremities. There was an ulcer of the right great toe, and pulsations could not be felt in the right dorsalis pedis and posterior tibial arteries. The arteries of the left lower extremity pulsated normally, except the left dorsalis pedis and posterior tibial arteries, where the pulsations were slightly reduced. Pulsations of the vessels of the hand were normal. Rubor, graded 1 to 2, of the toes of both feet was present when the feet were dependent, and pallor, graded 1 to 2, of the toes of both feet was present when the feet were elevated. There was intermittent glycosuria, and the glucose tolerance curve gave evidence of mild diabetes. A restricted diet was instituted. Fever was induced by giving typhoid vaccine intravenously and sulphur-in-oil intramuscularly. Improvement gradually ensued; there was complete relief of the pain while at rest, the ulcer healed, and the patient was dismissed in two months in satisfactory condition.

The second admission was March 14, 1931. During the interval the man's condition had been satisfactory. There was no change in the pulsations of the peripheral blood vessels. The ulcer had healed; there was an occasional twinge of pain in this area. The vasomotor response to induction of fever caused an average increase of 10° C. in the temperature of the toes. The patient was considered to be in satisfactory condition for lumbar sympathetic ganglionectomy, but he felt that he was getting along so well he wished to postpone this procedure.

The third admission was September 1, 1931. The ulcer had remained healed. Claudication appeared after the patient had walked two or three blocks, and his activities had been kept at about 50 per cent of normal. The feet were warm, growth of nails was normal, and there was no change in the pulsations of the peripheral arteries. The diabetes was well controlled by diet, and the case was considered to be an instance of compensated thromboangiitis obliterans.

The patient's fourth admission was May 11, 1932. At this time the pulsations of the posterior tibial and dorsalis pedis arteries of the right foot were intermittent and reduced. There was claudication in the right leg after walking one to three blocks. A small area of superficial phlebitis was present on this leg. There were no symptoms of diabetes. The diabetes continued to be controlled with dietetic restrictions. The patient was given four injections of typhoid vaccine for production of fever, with satisfactory effects on the claudication.

This is a typical example of slowly progressive thromboangiitis obliterans. Closure of the vessels was not so rapid but that collateral circulation could keep pace. The patient reacted extremely well to fever therapy. The ischemic ulcer healed rapidly, and objective evidence of improvement was noted in the circulation of the extremities.

The diabetes was mild and easily controlled. Much will depend on the willingness of the patient to continue with a restricted diet. Any excess of carbohydrates undoubtedly will make this patient more liable to cutaneous infection in the presence of the somewhat diminished circulation.

One additional case of thromboangiitis obliterans with diabetes has been observed at the clinic, and was reported by Adams[1] in 1930. Two ad-

ditional subjects, both Russian Jews, aged respectively 31 and 44 years, both with diabetes, were also thought to have thromboangiitis obliterans, but the clinical evidence was not sufficient to allow a positive diagnosis of thromboangiitis obliterans to be made; for that reason these cases have not been included in the present report.

Summary

Three Russian Jews and a man of English-American extraction, aged respectively 42, 47, 38, and 42 years, had thromboangiitis obliterans and diabetes mellitus. All were excessive smokers of cigarettes. Thromboangiitis obliterans in these subjects did not seem to be accentuated by the presence of diabetes mellitus.

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HUMAN CONSTITUTION:

A STUDY OF THE CORRELATIONS BETWEEN PHYSICAL ASPECTS OF THE BODY AND SUSCEPTIBILITY TO CERTAIN DISEASES *

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THERE is a saying: One man's meat is another man's poison. This will be recognized as a crude statement of the constitutional peculiarities and differences of various members of the human species, and is another indication of the penetrating insight of the folk-lore poet that precedes the slow march of science. Where science goes beyond the philosopher is in asking why and then proceeding to find the answer.

For the purposes of this study we may paraphrase the saying somewhat as follows: The more nearly two individuals resemble each other, the greater are their chances of developing the same disease. It is highly probable for instance that we are all exposed to tuberculosis at some time in our lives, yet only a certain proportion of us develop the disease. Among those that do there is often a general family resemblance. We go through life on very much the same routine of eating, working and sleeping, and some of us develop hypertensive disease and others acquire gall-stones, and some develop diabetes or cancer. A study of the constitutional factors in patients suffering from these diseases will often show underlying trends in them that bring them into rather close relationship with one another, so that Draper [1] has been able to delineate an ulcer race, a gall-bladder race, a pernicious anemia race. It is easy to ask why, but the answer lies buried so deep in the peculiarities of structural make-up, of chemical and metabolic processes, and of psychologic outlooks, that go to make up the total personality, that we are, even in the beginning, somewhat baffled by the magnitude of the problem of constitution.

The constitution of an individual may be defined as the sum-total of all his peculiarities and potentialities. Just how much is a matter of genes and how much is conditioned by early environment cannot be determined accurately. After birth the body increases in size only about 20 or 30 times, whereas the increase from the ovum to the mature fetus may be a matter of billions. The changes imposed upon the constitution after childhood are certainly minimal. The equipment with which an individual starts life, his fundamental constitution, is largely determined by heredity, and Pende [2] has likened constitution to a three-cornered pyramid the base of which

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From Blackburn Laboratory; the Department of Neurology, George Washington University; and the Department of Biology of the School of Hygiene and Public Health, Johns Hopkins University. Aided by a grant from the Josiah Macy, Jr. Foundation.

represents heredity and the three sides, respectively, the structural, the chemical and the psychological aspects of the personality.

The chemical side of the personality is probably the most important from the standpoint of general medicine, since immunities and susceptibilities to disease are determined largely in this manner, not to mention the various metabolic processes, endocrine activities and other organic reactions. The correlation of different constitutional peculiarities of the individual, his physical architecture and his personality trends, with his susceptibilities and resistances to disease, has formed the basis of a number of interesting studies. Three years ago I [3] reported some correlations between psychologic reaction type and susceptibility to certain diseases, and it is possible now to amplify the previous findings by demonstrating some correlations between body type and susceptibilities to the same diseases. For this purpose, the material, consisting of some 1260 autopsied cases, has been divided into four groups corresponding to Kretschmer's [4] classification, asthenic, athletic, pyknic and dysplastic. The asthenic type is seen in the long thin individual with poorly developed muscles and a narrow costal angle. The pyknic type has a short broad body with a round head, and, as Kretschmer calls it, a magnificent paunch. The athletic type lies in between. It is not particularly distinguished as a separate group, since there are various types of athletes extending from the distance runner to the weight thrower. As a rule, however, the proportions of the athlete are more harmonious than those of the other two groups. The dysplastic individual does not fit into any of the above three groups by reason of a combination of pyknic and asthenic traits, or because of abnormalities of body that stamp him as distinctly freakish. Conditions are similar, in the main, for women, although the pyknic type is often taller and larger all around than is the athletic.

The distribution of the patients is given in table 1, which shows the per-

TABLE I

Distribution of Physical Types in 1081 Cases

Asthenic	31.8%
Athletic	34.8%
Pyknic	14.9%
Dysplastic	13.8%
Unknown (no data)	4.7%
Total	100%

centage of individuals falling in each class. It will be noted that the asthenic and athletic are about equal in number, and the pyknics half as many, the dysplastics making up only about one-eighth of the total cases. Allowance must be made for this uneven distribution in comparing the ratios of the various diseases and lesions found in subsequent tables.

A comparison of the physical and psychological types is of interest and tends to confirm the findings of Kretschmer. (Figure 1.) The asthenic habitus is most often associated with the shut-in schizoid personality trend,

while the pyknic habitus and the extrovert, cycloid personality run together. Furthermore, there is quite a predominance of dysplastic individuals among the epileptics, and the paranoids show a rather even distribution among

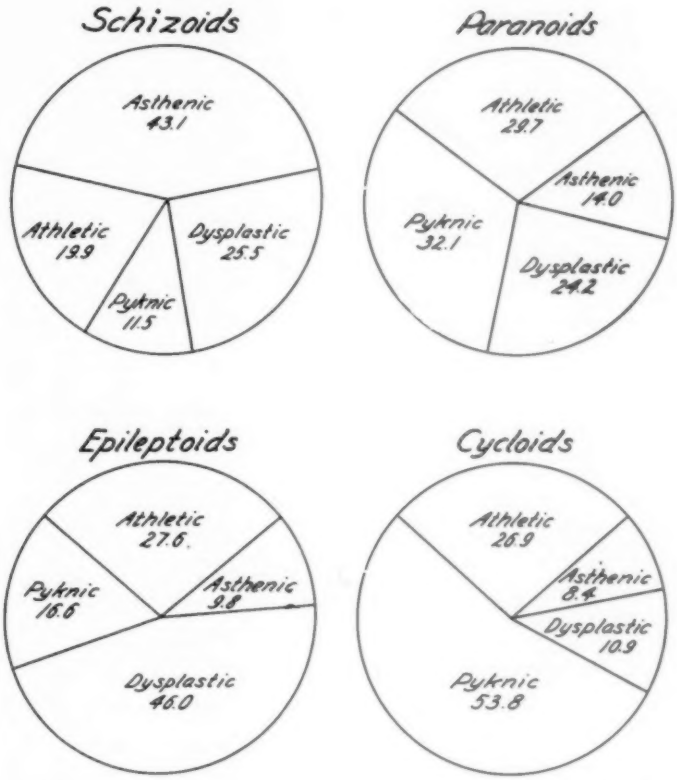

FIG. 1. Pie diagrams indicating physical make-up of the several psychologic classes.

classes with pyknics and athletics almost even. The small percentage of asthenics among the cycloids and of pyknics among the schizoids forms the reverse of the preceding picture. The individuals have been grouped according to body type and personality type alone, without regard to sex or race, and independently of the supposed cause of the psychosis. Our studies are showing that only minor differences are apparent between the symptomatic and the idiopathic psychoses as far as the personality types and body types are concerned, and that constitution transcends race, sex and age.

The distribution of a number of disease entities and of pathologic lesions is given in the accompanying table. (Table 2.) It must be called to mind that these percentages are not weighted, so that the relative incidence of the various lesions is not shown, but even so, certain findings stand out that indicate rather pronounced irregularities in the distribution of certain diseases. The high proportion of asthenics among the tuberculous, and of

TABLE II

Distribution of Disease Classes According to Body Types; Both Sexes, All Races

	Total Number of Cases	Percentage of Cases in Body Groups				
		Not differentiated	Asthenic	Athletic	Pyknic	Dysplastic
All cases 1260	1260	4.7	31.8	34.8	14.9	13.8
Cardio-vascular disease						
Chronic myocarditis, sclerosis, fibrosis	488	7.0	26.6	37.1	16.2	13.1
Coronary thrombosis, infarction, aneurysm, rupture	84	3.6	19.0	36.9	22.6	17.9
Pericarditis, acute, adhesive, chronic	88	2.3	34.1	35.2	11.4	17.0
Valvular disease, endocarditis, aortic and mitral stenosis	271	3.7	22.5	40.2	15.9	17.7
Cardiac syphilis	82	1.2	30.5	39.0	17.1	12.2
Aortic aneurysm, luetic or senile	32	—	9.4	43.7	34.4	12.5
Vascular thrombosis of aorta, lung, spleen, small intestine	192	3.1	20.3	39.1	20.8	16.7
Nervous diseases						
Cerebral hemorrhage	42	—	9.5	54.8	26.2	9.5
Cerebral thrombosis, infarction	316	5.4	23.4	40.2	16.1	14.9
Neurosyphilis	279	5.7	31.5	39.8	12.2	10.8
Encephalitis, meningitis	105	4.8	35.2	30.5	16.2	13.3
Pachymeningitis, subdural hematomata	95	6.3	27.4	45.2	5.3	15.8
Cerebral malformations, hydrocephalus, diffuse gliosis	75	5.3	22.7	30.7	12.0	29.3
Neoplastic disease						
Primary carcinoma all organs	127	7.0	27.1	38.8	12.4	14.7
Respiratory diseases						
Active tuberculosis	179	3.4	62.5	16.2	3.9	14.0
Healed tuberculosis	313	4.5	31.6	32.3	15.0	16.6
Lobar pneumonia and influenza	72	2.8	27.8	40.2	13.9	15.3
Bronchopneumonia	614	6.0	33.2	37.9	10.7	12.2
Pulmonary thrombosis, infarction, hemorrhage	126	1.6	19.8	38.9	24.6	15.1
Pulmonary abscess and gangrene	70	5.7	44.2	32.9	2.9	14.3
Bronchiectasis and syphilis	87	9.2	31.0	41.5	5.7	12.6

pyknics suffering pancreatic hemorrhage, is worthy of note, as well as the preponderance of intestinal disorders such as hernia, foreign bodies and intestinal gangrene among the athletic. A few other disorders, in which one special body type contains more than 40 per cent of the total lesions, may be mentioned. Cardiac valvular disease, aortic aneurysm, cerebral thrombosis and hemorrhage, nephritis, pyelonephritis, prostatic hypertrophy and malignancy, pituitary tumors, goiter and subdural hematoma occur in considerable numbers in the athletic type. Pulmonary abscess and gangrene as well as pulmonary and intestinal tuberculosis pick out especially the asthenic type, while pyknics comprise a rather high percentage of diabetics. The dysplastics are so few in number that they do not show any strikingly high percentages for any lesions.

Those diseases showing a relatively low distribution among certain body types may also be mentioned, taking 10 per cent as the dividing line. Few

TABLE II (*Continued*)

	Total Number of Cases	Percentage of Cases in Body Groups				
		Not differentiated	Asthenic	Athletic	Pyknic	Dysplastic
All cases 1260	1260	4.7	31.8	34.8	14.9	13.8
Gastrointestinal diseases						
Ulcers of stomach and intestine, enteritis	82	—	31.7	34.1	11.0	23.2
Carcinoma of stomach	30	—	33.3	30.3	13.3	23.3
Intestinal tuberculosis	89	1.1	75.3	13.5	1.1	9.0
Hernia, all types	14	7.1	14.3	71.4	—	7.1
Thrombosis, infarction, rupture, gangrene of small intestine	8	12.5	—	62.5	12.5	12.5
Foreign bodies	7	—	28.6	42.9	14.3	14.3
Chronic colitis	55	—	38.1	27.3	16.4	18.2
Cirrhosis of liver, mostly mild	289	3.1	26.6	34.4	18.3	17.6
Chronic cholecystitis with or without stones	264	5.3	29.5	35.7	14.0	15.5
Diabetes mellitus	17	5.9	11.8	29.4	41.1	11.8
Hemorrhagic pancreatitis	31	3.2	12.9	22.6	54.8	6.5
Carcinoma of biliary passages	15	6.7	20.0	46.7	13.4	13.4
Urogenital diseases						
Nephritis	74	4.1	32.4	40.5	6.8	16.2
Nephrosclerosis, fibrosis, infarcts, sclerosis, chronic inflammation of gonads	922	5.0	28.2	38.2	14.2	14.4
Pyelonephritis, abscess	119	7.6	30.3	41.1	10.9	10.1
Lithiasis	56	1.8	19.6	25.0	30.4	23.2
Cystitis	62	6.5	25.8	38.7	17.7	11.3
Prostatic hypertrophy	199	3.5	25.1	42.7	15.6	13.1
Carcinoma of prostate and bladder	22	4.5	31.8	40.9	4.5	18.2
Uterine fibroids	151	4.0	25.2	35.8	21.9	13.2
Ovarian cysts	83	2.4	34.9	32.5	18.1	12.0
Urogenital malformations	58	1.7	27.6	25.9	12.1	32.8
Endocrine diseases						
Pituitary tumors	52	3.8	15.4	51.9	15.4	13.5
Goiter	105	3.8	27.6	40.0	14.3	14.3
Persistent thymus	168	—	31.0	36.3	11.3	21.4
Adrenal hemorrhage	16	6.3	37.5	37.5	6.3	12.5
Adrenal adenomata and hypernephromata	45	—	31.1	28.9	15.6	24.4

aortic aneurysms and cerebral hemorrhages occur in asthenic individuals. Pulmonary abscess and gangrene and bronchiectasis, as well as tuberculosis of the lungs, are rare in pyknics; few cases of nephritis, of prostatic carcinoma, of pachymeningitis and of adrenal hemorrhage occur in pyknics. The dysplastic group is represented by relatively few instances of hernia and of hemorrhagic pancreatitis. The athletic group has a fairly large proportion of all the different lesions both on account of the size of the group and because it stands more or less in mid-position between contrasting body types.

In order to contrast the distribution of various groups of diseases among the four body types, figure 2 has been constructed. The relatively even distribution of the acute infectious disorders (compare with table 1) lends added weight to the differences observed in the other categories, since the

acute infections as such are more strictly conditioned by external factors (microörganisms) than by constitutional traits.

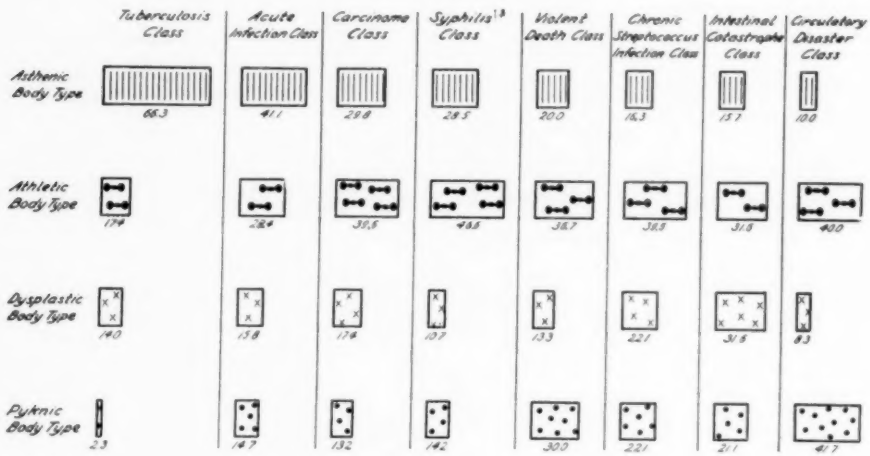

FIG. 2. DISEASE TYPE AND BODY TYPE

Percentage of individuals of different body types having a specified disease type. Both sexes and all races are included, but only those individuals that have been differentiated as to both body type and disease type. Individuals listed as having more than one disease are classified in only the more important group. The order of importance of disease groups has been taken to be the following: Tuberculosis takes precedence over all other groups; syphilis next, then carcinoma, circulatory disease, streptococcal infections, intestinal disease, violent deaths, and acute infections, in the order named. Therefore, tuberculosis and syphilis occurring together would be counted only in the tuberculosis group, as would tuberculosis and circulatory disease. Syphilis and violent death would be classified only under syphilis. The second disease in each case is ignored.

Comparing the distribution of separate disease entities according to physical type with the distribution of the same diseases according to psychologic type brings out the noteworthy fact that distinctions are sharper in the latter. This would seem to indicate that the personality of the individual is more closely linked with his immunologic attributes than is his physical build.

Finally, from a study of the combined figures, a reconstruction of the constitutional pyramid of Pende, certain constellations may be picked out as contrasting examples of human biotypes. The asthenic-tuberculous-schizoid contrasts with the pyknic-angiopathic-cycloid, and both of them with the dysplastic-exudative-epileptoid, while another less striking example is the athletic-paranoid group showing decided tendencies toward malignant disease and chronic streptococcus infections.

The necessity for a study of the patient as a whole is again emphasized. This contribution shows that there are certain diseases that show a predilection for one physical type or another, just as the former study showed that some diseases fell more heavily on one psychological type than another. Rather definite relationships between mental and physical types complete the

triangle of constitution, and it is possible to erect certain constellations comprising all three facets of the individual's total personality.

The author is indebted to Miss Marjorie Gooch, Associate in the Department of Neurology, George Washington University, for the statistical handling of the data.

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PAROXYSMAL VENTRICULAR TACHYCARDIA:

AN ETIOLOGICAL STUDY WITH SPECIAL REFERENCE TO THE TYPE*

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PAROXYSMAL TACHYCARDIA of ventricular origin is a cardiac disorder which, because of its severity, deserves and has received much study. There are, however, questions as to its mechanism and etiology that are still unanswered. This is our warrant for reporting three cases and for recording the results of a survey of the literature on this subject. We have endeavored to get facts as to the causes that excite its outbreak as well as to the nature of the heart disease which underlies this condition. This survey may also be helpful in an attempt to correlate these findings with a classification of the various types of the arrhythmia, and thus bring about a closer relationship to the exciting causes and foundation etiological factors.

CASE I

A boy, A. D., 16 years of age, presented himself at the Central Free Dispensary, January 26, 1930, complaining of spells of tachycardia for the previous six months. The duration of a spell varied from a few days to as long as three weeks. His strength and endurance were poor. He had noticed shortness of breath on exertion for three months, and afternoon swelling of his feet and ankles for one month. He gave no history of the usual childhood diseases except measles. The only other illness was rather frequent sore throat for the past three months.

Examination revealed a boy 5 feet 7 inches in height, weighing 145 pounds. His face was flushed. The teeth were in good condition. The tonsils were small, slightly injected and contained a few crypts. No pus could be expressed. The thyroid was soft, and uniformly enlarged to a slight degree. The lungs were normal. There was a diffuse heaving impulse of the heart that was visible over an area measuring 7 by 15 cm. and extending from the second to the sixth interspaces immediately to the left of the sternum. The left heart border was found to be 13 cm. to the left of the midsternal line in the sixth interspace; the right border 3 cm. to the right of the midsternal line in the fifth interspace. The heart rate was 125 and regular. The pulse was weak, but every beat was felt at the wrist. A gallop rhythm was heard over the entire precordium, but was loudest 2 cm. to the right of the sternum in the fourth interspace. Three distinct impulses were imparted to the hand placed over the heart. No thrill was palpable, and no murmurs were heard. The examination of the abdomen revealed nothing pathological. The reflexes were normal. There was no evidence of edema, cyanosis, or dyspnea.

The blood pressure was 116/96 mm. of mercury. Blood counts and hemoglobin estimations were normal. The blood Wassermann was negative. The urine contained albumin two plus; no casts. The basal metabolic rate was plus 15 and plus 8.

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From the Medical Departments of Rush Medical College of the University of Chicago and the Presbyterian Hospital of Chicago.

Roentgen-ray examination with a seven foot plate showed the greatest transverse diameter of the chest to be 29.6 cm., and of the heart, 20.5 cm. The greatest width of the supracardiac shadow at the level of the aortic knob was 7 cm. The base of the heart measured 16.5 cm. The lungs were normal. The electrocardiograms are shown in figures 1, 2, and 3.

Diagnosis. Paroxysmal tachycardia of right ventricular origin; cardiac dilatation and hypertrophy; heart disease of unknown etiology.

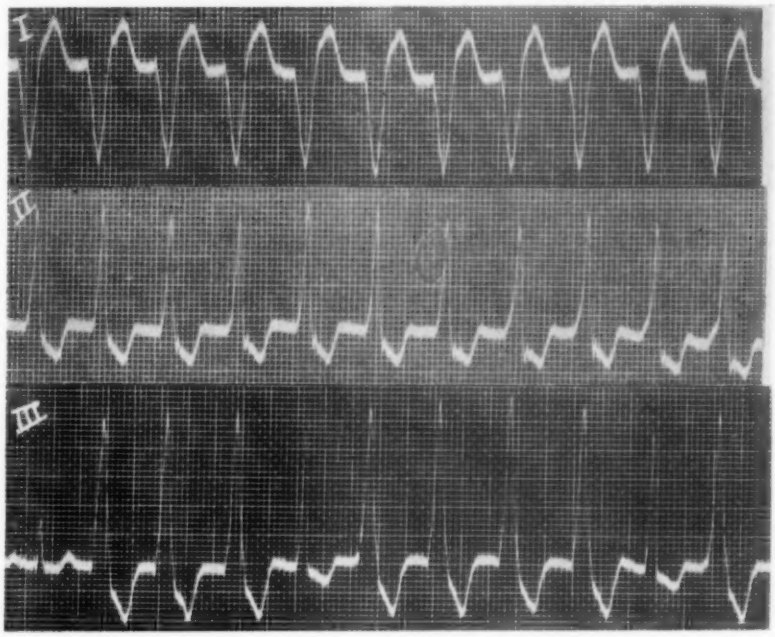

FIG. 1. *Case I.* (January 26, 1930.) Boy, A. D., 16 years old. Rate 125. Paroxysmal tachycardia of right ventricular origin. Note one normal beat in Lead III. In all tracings 1 cm. = 1 mv. and time interval = .04 second. *Note:* In Lead III, 0.5 cm. = 1 mv. This is the only exception.

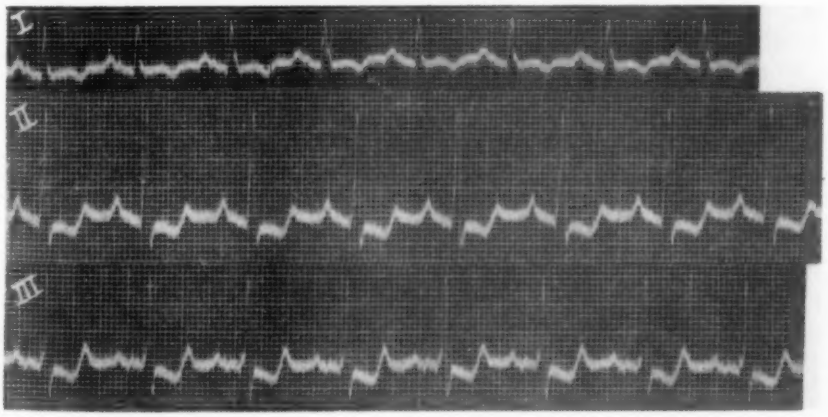

FIG. 2. *Case I.* A. D. (March 14, 1930.) Rate 78. Normal rhythm after quinidine.

Etiology. There was none obtainable either for the attacks or for the underlying heart disease. There was no history of previous disease, strain, or excesses which could account for the condition.

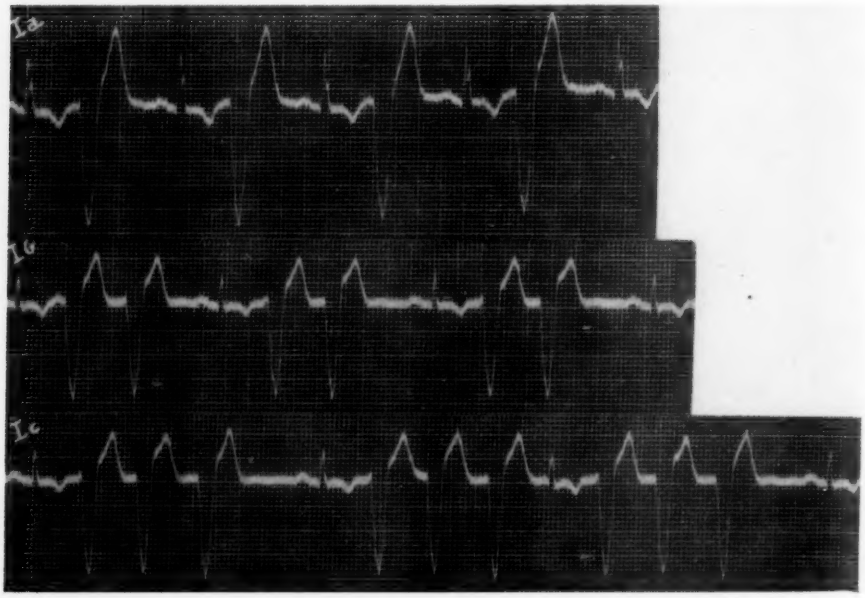

FIG. 3. *Case I.* A. D. All strips are of Lead I. Extrasystoles observed throughout subsequent observation.

Treatment and Course. Vagal and orbital pressure were without effect. The rapid tachycardia was temporarily controlled by quinidine sulphate, given in gradually increased dosage up to 12 grains per day. Examinations, when the rate was normal, showed that the heart had not changed in size. The left border was 11 cm. to the left of the mid-sternal line in the sixth interspace, and 3 cm. to the right of the mid-sternal line at the fifth interspace. A seven foot roentgen-ray plate taken during normal rate, gave essentially the same measurements as were found during the spell of tachycardia.

The patient continued to take the quinidine, but within a month the spells of tachycardia returned. Quinidine was given up to 42 grains per day, but the rhythm never returned to normal. Runs of right ventricular extrasystoles persisted in spite of digitalis, quinidine, potassium iodide, atropine, strychnine, and morphine. A rapid regular sinus rhythm could be established by exercise, but it would persist for only one or two minutes. At present the patient is at home confined to bed under the care of an outside physician. His condition is very poor, and the prognosis bad.

CASE II

A man, A. L., 59 years of age, entered the Presbyterian Hospital of Chicago, March 31, 1930, because of a recurring biliary fistula. He had had an operation in 1918 for gangrenous gall-bladder with stones. The gall-bladder was not removed. The wound drained for six months and gradually closed. In 1920 the wound broke open and discharged for a few weeks. The fistula then remained closed until one year ago (1929) when it reopened and has discharged ever since. Slight jaundice, chills, and fever developed.

Additional past history disclosed the fact that the patient had had rheumatic fever twice (at 19 and 36 years of age) and had been told that there was some heart involvement each time. Recently he had had some dyspnea and heart consciousness on exertion.

Cholecystectomy was performed April 2, 1930, by Dr. Arthur Dean Bevan who found a common duct obstruction of unknown cause. Convalescence was prolonged due to persistent discharge and to delayed healing of the incision. May 12, 1930, the patient was allowed to be up in a wheel chair for one hour.

On May 13, 1930, the patient on waking stated that he did not feel well and complained of marked tachycardia. Examination revealed a regular pulse rate of 200. No murmurs were heard. The left heart border was 11 cm. to the left of the midsternal line in the fifth interspace. The right heart border was substernal. Orbital and vagal pressure were without effect. The electrocardiograms are shown in figures 4 and 5. Note the coronary T-wave in figure 5. There was no history of

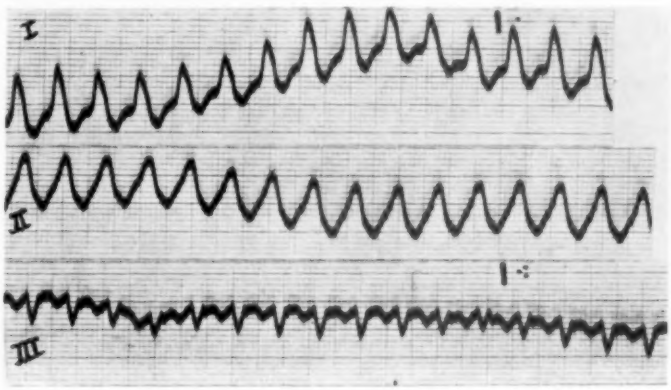

FIG. 4. *Case II.* (May 13, 1930.) Man, A. L., 59 years old. Rate 187. Paroxysmal tachycardia of left ventricular origin.

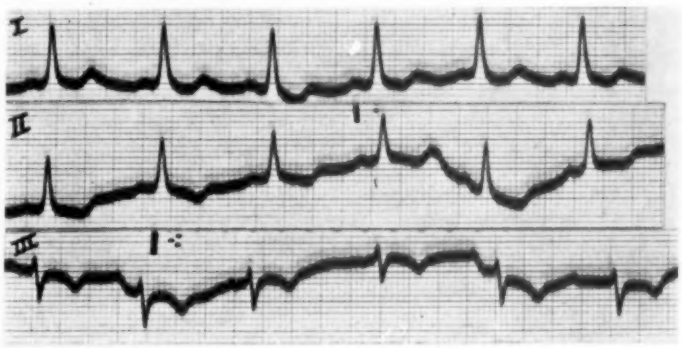

FIG. 5. *Case II.* A. L. (May 14, 1930.) Rate 83. Normal rhythm, probable coronary T-wave.

angina. The attack suddenly ended after about three hours. Extrasystoles persisted for about 30 minutes longer. There were no recurrences in the hospital and the patient was discharged June 14, 1930. Within the following six weeks there were two similar but shorter attacks, each brought on by over-exertion and relieved by rest.

Cardiac Diagnosis. Paroxysmal tachycardia of left ventricular origin; history of rheumatic heart disease; probable coronary sclerosis.

Etiology. The attacks of paroxysmal tachycardia may be attributed to coronary sclerosis. The heart disease is attributed to rheumatic fever. Chronic biliary infection may have been a contributory cause.

Treatment and Subsequent Course. The patient was seen frequently and remained free from attacks for four months. In December 1930, he developed acute cardiac decompensation with edema, cyanosis, dyspnea and a total arrhythmia. The electrocardiogram confirmed the diagnosis of auricular fibrillation. At present the fibrillation persists, and the decompensation is slowly responding to digitalis and rest.

Case III

A man, H. B., 42 years of age, was first seen in the Central Free Dispensary in April 1918. His complaints at that time were palpitation and precordial pain on exertion, joint pains, insomnia and anorexia. The past history revealed measles in childhood, frequent attacks of tonsillitis, chancre in 1905, and gonorrhea in 1910. Four blood Wassermanns were reported to be negative.

Physical examination revealed rather large tonsils. The left border of the heart was 14 cm. to the left in the fifth interspace; the right heart border was substernal. There were occasional extrasystoles. A soft murmur was heard over the lower end of the sternum. Lungs, abdomen, and reflexes were normal.

Laboratory examinations showed the hemoglobin 85 per cent; white blood cells 12,000; urine normal; blood Wassermann four plus; blood pressure 130/70 mm. of mercury. Roentgen-ray showed an enlarged heart with a tendency towards a mitral configuration.

Diagnosis (April 1918). Syphilis with cardiac hypertrophy and dilatation. Probable syphilitic heart disease.

Treatment. Mercury and iodide for one year; then 0.2 gm. neosalvarsan intermittently for two years. The patient then disappeared until June 1925, when he returned complaining of epileptiform seizures and precordial pain. The record of the physical examination at that time is not available.

Diagnosis (June 1925). Epileptiform seizures, coronary occlusion, mitral stenosis, auricular fibrillation, syphilis. The electrocardiogram showed a coronary T-wave. (See figures 6 and 7.)

Treatment. Digitalis, neosalvarsan, iodides, and mercury. There was a marked improvement—the blood Wassermann became negative, regular sinus rhythm was restored, the patient's heart was well compensated, and the epileptiform seizures disappeared.

Subsequent Course. The patient returned in June 1928, with jaundice and attacks of pain in his gall-bladder region. Cholecystectomy was done by Dr. Harry Oberhelman who found four stones in the gall-bladder. Recovery was rapid. In July 1928, severe left chest pains developed. At this time he gave the first history of spells of tachycardia. These were associated with the attacks of pain in his left chest. He was seen during an attack when the pulse rate was 178 and regular. The heart was considerably enlarged; during this period of tachycardia no murmurs were heard. The electrocardiogram showed a tachycardia of left ventricular origin. Treatment consisted of rest, nitroglycerin, and digitalis. The spells were of rather frequent recurrence for one year. Quinidine was not used. After August 1929, the attacks became less frequent but the pain persisted. Examination at that time revealed aortic regurgitation as well as mitral stenosis. The presence of an Austin Flint murmur was considered but ruled out. The heart had become considerably larger. The left heart border was in the anterior axillary line; the right heart border 3 cm. to the right of the right sternal margin. In January 1930, the heart was compensated and the pa-

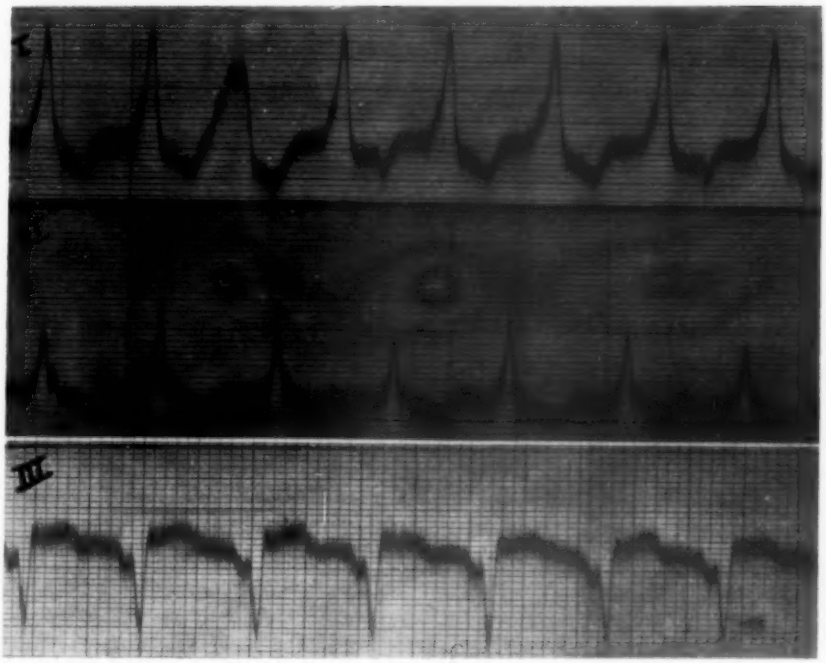

FIG. 6. *Case III.* (July 9, 1928.) Man, H. B., 42 years old. Rate 143. Paroxysmal ventricular tachycardia of left ventricular origin. Retrograde P-waves in Lead I.

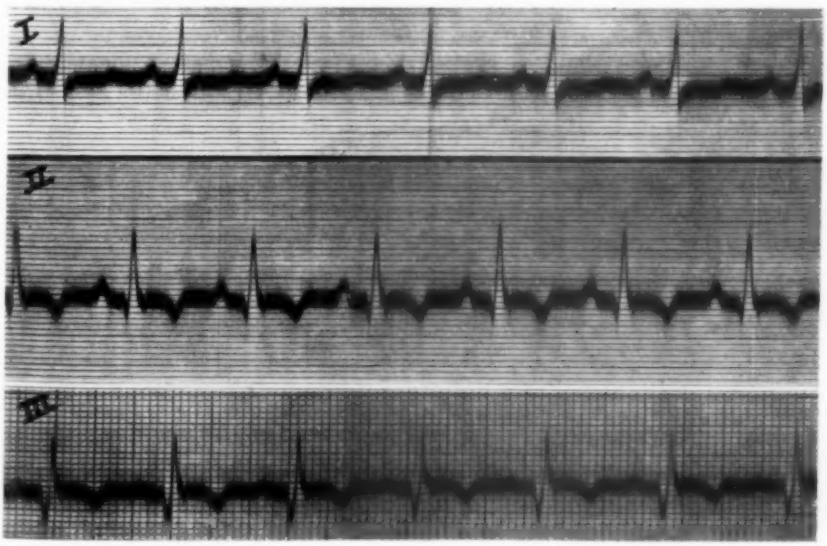

FIG. 7. *Case III.* H. B. (August 25, 1929.) Rate 97. Normal rhythm with coronary T-wave.

tient was getting along fairly well with 15 drops of tincture of digitalis three times a day, 1/100 grain of nitroglycerin as needed, and 3/4 grain of luminal twice a day.

At present the paroxysms of tachycardia still occur, but are infrequent and of short duration; extrasystoles are constantly present.

The exciting cause for the attacks of paroxysmal tachycardia would seem to be coronary occlusion; the etiology of the heart disease is syphilis, rheumatism, and chronic tonsillitis.

During the past few years, several reviews of this arrhythmia have appeared. These reports are concerned chiefly with only one phase of the subject. No one has tabulated all of the proved cases and analyzed the exciting causes for the onset of the attacks in relation to the underlying heart disease. We have made such a survey and have found some valuable information by

TABLE I

Right Ventricular Type. (23 Cases.)

Ref. No.	Our Case No.	Age	Sex	Valve Disease	Etiology for Attacks	Etiology for Heart Disease	Degree of Heart Disease	Outcome
1	1	Mid.	M	Murmur	Exertion; onset after box fell on leg	Gouty arthritis Influenza	Mod.	L
5	5	20	M	—	Diphtheria	Diphtheria	Mild	L
7	11	42	M	—	None	Strept. infections	Mod.	L
10	19	49	M	Aortic	Coronary occlusion	Syphilis Rheumatic fever	Mod.	L
12	21	39	F	None	None	None	None	L
13	22	21	F	—	None	Pneumonia Diphtheria Tonsillitis	Mod.	L
17	27	35	F	None	None	Dry pleurisy (In bed 1 yr.)	None	L
22	34	60	M	—	None	None	Adv.	L
22	35	18	M	Murmur	None	Rheumatic fever	Adv.	L
23	38	63	M	Aortic	Digitalis	Arteriosclerosis	Mod.	D*
23	40	60	F	Murmur	Digitalis	None	Adv.	D
29	48	25	M	—	Focal myocarditis Early coronary arteritis	Abscessed teeth Infected tonsils	Adv.	D*
30	49	53	M	—	Exertion	None	Mod.	L
34	58	18	M	—	None	Pneumonia twice	None	L
35	59	?	M	Mitral	Coronary disease	Hypertension	Adv.	L
38	62	50	F	—	None	Rheumatism	Mild	L
42	71	53	M	—	Coronary thrombosis	None	Mod.	L
44	74	17	?	—	None	None	Mild	L
45	78	51	M	—	Coronary thrombosis	None	Adv.	D*
47	80	20	M	None	None	None	None	L
47	81	23	M	None	None	None	None	L
51	88	17	M	—	Undue excitement	None	None	L
52	89	16	M	—	None	None	Adv.	L

* Autopsy.

correlating the individual case histories with a classification of the types of this arrhythmia.

We have found reference to 149 cases which, with our own, make a total of 152 cases. Of these 152 cases, 56 [5, 7, 9, 21, 45, 53–75, 81] were discarded as unproved because of questionable or absent electrocardiograms or because of insufficient historical data. The data of the remaining 96 cases were studied with reference to age, sex, etiology of the attacks, etiology of the associated heart disease, the degree of heart disease, the type of arrhythmia,

TABLE II

Idio-Ventricular Type. (23 Cases.)

Ref. No.	Our Case No.	Age	Sex	Valve Disease	Etiology for Attacks	Etiology for Heart Disease	Degree of Heart Disease	Outcome
3	3	45	M	Mitral Aortic	Coronary disease	None	Adv.	D*
4	4	48	M	Murmur	Rheumatism	Rheumatism	Mod.	L
5	6	Young	M	—	None	None	None	L
5	7	Young	M	—	Exertion	None	None	L
6	9	50	M	—	Poor blood supply	None	Mod.	L
7	10	62	M	—	None	Strept. infections	Mod.	L
9	15	23	F	Mitral	Terminal digitalis	Rheumatic fever	Adv.	D
10	18	53	F	—	Coronary occlusion	None	Adv.	D
19	30	52	M	—	Coronary occlusion	Arteriosclerosis	Adv.	L
23	37	50	F	Mitral	Digitalis	Hypertension	Adv.	L
25	42	46	M	Aortic?	Coronary occlusion	None	Mod.	L
26	43	32	M	—	None	Rheumatism	Mod.	L
33	55	61	M	—	Coronary disease	Arteriosclerosis Asthma Influenza	Adv.	D
33	57	19	M	—	None	None	None	L
39	63	12	M	—	None	None	Mild	L
40	64	71	M	—	Digitalis Ouabaine	Asthmatic bronchitis	Adv.	L
41	70	74	M	Murmur	Digitalis	Arteriosclerosis Hypertension	Adv.	D
43	72	50	M	—	Digitalis Coronary thrombosis	Arteriosclerosis Hypertension	Adv.	D*
45	75	41	F	—	None	Influenza	Mild	L
50	85	47	M	—	Digitalis Coronary thrombosis	Hypertension	Mod.	D*
50	86	54	M	—	Coronary occlusion Digitalis?	Hypertension	Adv.	D
79	92	16	F	—	None	None	None	L
81	95	52	M	—	Exertion	Coronary disease Arteriosclerosis	Mild	L

* Autopsy.

factors which might influence the type of arrhythmia, the outcome, and the revelations and bearings of autopsy findings. It may be objected that in a few instances the type of arrhythmia was determined from only one lead. These few instances, however, are not enough to make a significant differ-

ence in the results, so we feel justified in adding them to our series. Another criticism might be made regarding the inclusion of the alternating bidirectional type of the arrhythmia. This type is by some regarded as an alternating bundle branch block due to excessive digitalis administration.[29] The type is usually considered as ventricular. We include it for this reason, although we do not consider it to be a true paroxysmal ventricular tachycardia.

We are adhering to the old terminology with regard to the type of the arrhythmia. When QRS is upright in Lead I and inverted in Lead III we call this the left ventricular type.

The tabulation of our findings, according to types, follows:

A significant observation is that excessive digitalis administration is held responsible for 35 (36 per cent) out of the total of 96 cases of all types of this arrhythmia. The association is greater in the left ventricular and idio-ventricular types than in the right. We believe this is due to the fact that digitalis makes the left ventricle work harder than the right (due to the

TABLE III

Left Ventricular Type. (35 Cases.)

Ref. No.	Our Case No.	Age	Sex	Valve Disease	Etiology for Attacks	Etiology for Heart Disease	Degree of Heart Disease	Outcome
2	2	49	M	Murmur	Coronary disease	Syphilis	Mod.	L
6	8	21	M	Aortic Mitral	Poor blood supply	Rheumatism Sore throats	Adv.	L
7	12	38	M	—	—	Strept. infections	Adv.	L
8	13	59	M	Mitral	Terminal	Hypertension	Adv.	D*
8	14	63	M	Aortitis	—	Syphilis	Adv.	L
10	16	53	M	—	Coronary occlusion	Syphilis Hypertension	Adv.	D*
10	17	58	M	—	Coronary occlusion	Arteriosclerosis Hypertension	Adv.	D
11	20	22	M	—	Exertion	—	None	L
14	23	58	M	—	Quinidine	—	Adv.	L
15	24	20	M	—	None	None	Mod.	L
15	25	57	M	Aortic	—	Rheumatism Pleurisy	Adv.	L
16	26	?	?	—	Quinidine	—	—	L
20	31	49	M	—	—	Teeth	Mod.	L
22	33	20	F	—	—	—	Adv.	L
22	36	58	F	Mitral	—	Syphilis Hyperthyroidism	Adv.	L
23	39	80	F	—	Digitalis	Chronic tonsillitis	Adv.	L
24	41	47	F	—	—	—	None	L
27	44	45	F	—	—	—	Adv.	L
28	47	71	M	—	Digitalis Coronary thrombosis	Arteriosclerosis	Adv.	D*
32	52	48	M	Mitral	Exertion	Head colds Pharyngitis	Mod.	D (Pneumonia)

TABLE III—(*Continued*)

Ref. No.	Our Case No.	Age	Sex	Valve Disease	Etiology for Attacks	Etiology for Heart Disease	Degree of Heart Disease	Outcome
33	53	52	F	Mitral Tricuspid	Digitalis	Rheumatic fever	Adv.	D
33	54	64	M	—	Digitalis	Arteriosclerosis Asthma	Adv.	D
33	56	41	M	—	Nicotine poisoning	Tobacco	Mod.	L
36	60	54	M	—	Coronary thrombosis	Rheumatism	Adv.	D*
40	65	63	M	Mitral	Coronary disease (?)	—	Adv.	L
41	69	39	F	Aortic Mitral	Digitalis	Rheumatic fever	Adv.	D*
45	76	53	M	—	Coronary disease	—	Adv.	L
45	77	55	M	—	Exertion Coronary thrombosis	Hypertension	Adv.	D*
46	79	46	M	Aortitis	Exertion Coronary disease	Syphilis	Adv.	D*
48	82	45	F	—	Pressure inf. vena cava Megacolon	—	Mild	D*
49	84	73	M	—	Digitalis Coronary occlusion	Arteriosclerosis	Adv.	D
52	90	59	M	—	Coronary disease	Rheumatic fever Biliary infection	Adv.	L
52	91	42	M	Aortic Mitral	Coronary disease	Syphilis Chronic tonsillitis	Adv.	L
80	93	66	F	—	Coronary disease	Coronary disease Arteriosclerosis Hypertension	Adv.	D
81	95	31	M	—	Exertion Excitement	Scarlet fever Pneumonia	Mod.	D

* Autopsy.

fact that the resistance opposed to the complete emptying of the ventricles is greater for the left ventricle than for the right). In the presence of disease, the left ventricle and septum would therefore undergo a disproportionate strain. Digitalis intoxication in the presence of advanced heart disease is held accountable for 100 per cent of the cases showing the alternating bidirectional type.

No exciting causes could be determined for the attacks in 28 cases. In this group the right ventricular type predominates, 12 in number, while there are only nine in the larger group of the left ventricular type. This seems significant, especially in view of the association of the right ventricular type with a lower age incidence and a lesser degree of heart damage than in the left. Within the groups according to types, 12 cases (52 per cent) of the right ventricular type gave no exciting causes, and only 9 cases (25 per cent) of the left ventricular type failed to be associated with exciting factors for the attack. This observation seems to be associated with a better prognosis for the right ventricular type.

Coronary disease is associated with the onset of the attacks in 27 cases (28 per cent). The frequency of occurrence is greater in the left than in

TABLE IV

Alternating Bi-directional Type. (21 Cases.)

Ref. No.	Our Case No.	Age	Sex	Valve Disease	Etiology for Attacks	Etiology for Heart Disease	Degree of Heart Disease	Outcome
18	28	50	F	Aortitis	Digitalis	Syphilis Aneurysm	Adv.	D*
18	29	61	F	Mitral	Digitalis	Rheumatic fever	Adv.	D*
21	32	54	F	—	Digitalis	Hypertension	Adv.	D
28	45	64	F	—	Digitalis	None	Adv.	D
28	46	44	F	—	Digitalis	Exophthalmic goiter	Adv.	D
31	50	77	M	—	Digitalis Coronary disease	Arteriosclerosis	Adv.	D*
31	51	66	M	Aortic	Digitalis	Syphilis	Adv.	D
37	61	76	M	—	Digitalis Coronary thrombosis	Arteriosclerosis	Adv.	D*
41	67	53	F	None	Digitalis	Hypertension	Adv.	D*
41	68	42	F	Murmur	Digitalis	Arteriosclerosis Hypertension	Adv.	D
43	73	45	M	—	Digitalis	Chronic sore throat	Adv.	D
49	83	49	M	Aortic	Digitalis	Syphilis	None	D*
50	87	65	M	Aortic	Digitalis	Syphilis; obesity	Adv.	D
41	66	59	M	Aortic Mitral	Digitalis	None	Adv.	D*
23	39	80	F	—	Digitalis	Chronic tonsillitis	Adv.	L
28	47	71	M	—	Digitalis Coronary disease	Arteriosclerosis	Adv.	D*
41	69	39	F	Aortic Mitral	Digitalis	Rheumatic fever	Adv.	D*
41	70	74	M	Murmur	Digitalis	Arteriosclerosis Hypertension	Adv.	D
43	72	50	M	—	Digitalis Coronary thrombosis	Arteriosclerosis Hypertension	Adv.	D*
50	85	47	M	—	Digitalis Coronary thrombosis	Hypertension	Mod.	D*
82	96	40	M	—	Digitalis	Rheumatic fever	Adv.	L

* Autopsy.

the right ventricular type, which conforms to the generally recognized fact that coronary disease involves the left ventricle more often than it does the right ventricle.

Exertion is associated with the onset in 9 cases (9 per cent). Most of these, five cases, were of the left ventricular type. This is in contrast to the observation that in athletes the right ventricle hypertrophies. This difference may be due to the fact that exertion on the part of an untrained person causes strain on the left ventricle, whereas in long continued effort the strain is on the right ventricle.[79] Concomitant coronary disease may have helped determine the type in two of these cases.

Upon arranging this information according to groups, the following relationships are shown:

TABLE V

Summary of Exciting Causes for the Onset of Attacks

Number of Cases	Exciting Cause	Types			
		Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
96 total		23 cases	23 cases	35 cases	21 cases
35 (36%)*	Digitalis	2 (9%)†	6 (26%)†	6 (17%)†	21 (100%)† includes 6 duplicated types
28 (29%)*	None	12 (52%)†	7 (30%)†	9 (26%)†	5 (24%)† includes 3 duplicated types
27 (28%)*	Coronary disease	5 (22%)†	8 (35%)†	13 (37%)†	0
9 (9%)*	Exertion	2 (9%)†	2 (9%)†	5 (14%)†	0
8 (8%)*	Pulmonary disease	3 (13%)†	2 (9%)†	3 (9%)†	0
3 (3%)*	Toxic and infectious	1 (4%)†	1 (4%)†	1 (3%)†	0
3 (3%)*	Nervous	2 (9%)†	0	1 (3%)†	0
2 (2%)*	Quinidine	0	0	2 (5%)†	0

* % refers to total number of cases considered.
† % refers to total number of cases considered of this type.

Note: The correct percentage and relationship of terminal cases could not satisfactorily be determined. Two cases reported by Dieuaide and Davidson [9] are probably representative of the type to be expected under these circumstances. These included one left ventricular and one idio-ventricular type.

Pulmonary disease is associated with the onset in eight cases. While this group is small it seems worth while to call attention to the fact that within the sub-groups, three or 13 per cent were of the right type, whereas three in the larger group of the left type constitute only 9 per cent, showing a higher incidence in the right type. The strain on the right heart as a result of pulmonary circulation embarrassment may have been a factor in the production of the right ventricular type of the arrhythmia in these cases.

Nervous factors influencing the onset occurred more often in the right type than in the left. Quinidine was associated with the onset in two cases of the left type.

These results differ from those published by Strauss [49] largely because of the recent addition of quite a number of cases to the literature. The most notable differences are the shifting of arteriosclerotic heart disease and coronary disease to first and third places respectively instead of second and fourth. Hypertensive heart disease is displaced from first to fifth place. The percentage of rheumatic heart disease remains the same but moves from third to sixth place. The percentage of syphilitic heart disease is doubled, but its relative position of importance remains the same. The percentage

of undetermined kind of heart disease is greatly increased from 11 per cent to 32 per cent. We have listed (table 7) 13 cases (14 per cent) associated with no demonstrable heart disease, which agrees with Strauss' report of 11 cases or 15 per cent.

TABLE VI

Kinds of Associated Heart Disease with Relation to Type of Arrhythmia

Number of Cases	Kinds of Disease	Types			
		Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
96	(163 reported)	23 cases	23 cases	35 cases	21 cases
33 (34%)*†	Arterio-sclerotic	6 (26%)‡	10 (43%)‡	14 (40%)‡	6 (29%)‡ includes 4 duplicate types
31 (32%)*	Not determined	11 (48%)‡	9 (39%)‡	9 (26%)‡	2 (10%)‡
29 (30%)*	Coronary disease	5 (21%)‡	4 (17%)‡	13 (37%)‡	5 (24%)‡ includes 3 duplicate types
20 (21%)*	Toxic and infectious	6 (26%)‡	4 (17%)‡	9 (26%)‡	1 (5%)‡
17 (18%)*	Hypertensive	1 (4%)‡	5 (22%)‡	5 (14%)‡	6 (29%)‡
16 (16%)*	Rheumatic	4 (17%)‡	3 (13%)‡	6 (17%)‡	5 (24%)‡
14 (15%)*	No heart disease	6 (26%)‡	4 (17%)‡	3 (9%)‡	1 (5%)‡
11 (12%)*	Syphilitic	1 (4%)‡	0	6 (17%)‡	4 (18%)‡
2 (2%)*	Thyrotoxic	0	0	1 (3%)‡	1 (5%)‡
1 (1%)*	Obesity	0	0	0	1 (5%)‡

* % refers to the total number of cases considered in this series.
† includes 29 cases of coronary disease listed below.
‡ % refers to the total number of cases within the group of this type.

In addition, we call attention to the fact that hypertensive, arteriosclerotic, coronary, and syphilitic heart diseases are associated with a higher percentage of cases of the left ventricular type than of the right. This is expected since in all of these instances the heart damage is usually greatest in the left ventricle. Rheumatic heart disease does not live up to expectations in that the percentages of the left and right ventricular types are equal. We expected to find more of the right ventricular type since mitral disease is more common than aortic disease, and in mitral disease the stress and strain is greatest in the right ventricle. This exception affords an excellent opportunity to apply, to paroxysmal ventricular tachycardia, F. N. Wilson's[81] recent contributions to heart physiology, in which he gives reasons for reversing our present conception of right and left bundle branch block. This is an explanation worthy of serious consideration, and could be readily accepted were it not for the fact that all of the other kinds of heart disease are associated with the types which agree with our present conception of paroxysmal left and right ventricular tachycardia. We will return to this discussion when we consider the autopsy findings in the various types and draw our final conclusions at that time. For the present we will continue our reasoning while accepting the classical terminology and offer as an ex-

planation for this unexpected association the thought that after all, in mitral disease, the disease is on the left side of the heart. Later we shall show that in valvular disease, the type of arrhythmia which occurred was influenced by co-existing disease.

Toxic and infectious diseases of the heart also have an equal percentage of both types.

Attention is called to the fact that in 31 cases, where the kind of heart disease was not determined, there is a higher percentage of the right ventricular type than of the left.

TABLE VII

Degree of Myocardial Damage Associated with Types

Total Number	Degree of Damage	Types			
		Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
102 includes 6 duplicates		23 cases	23 cases	35 cases	21 cases
14 (14%)	None	6 (26%)	4 (17%)	3 (9%)	1 (5%)
7 (7%)	Slight	3 (13%)	3 (13%)	1 (3%)	0
20 (20%)	Moderate	7 (30%)	6 (26%)	6 (18%)	1 (5%)
61 (61%)	Advanced	7 (30%)	10 (44%)	25 (71%)	19 (90%)
47 (47%)	Deaths	4 (17%)	8 (33%)	15 (43%)	19 (90%)

The clinical evidence shows that the degree of myocardial damage is greater in the left ventricular type than in the right, and greatest in the alternating bi-directional type. Likewise the percentage of deaths was more in the left ventricular type than in the right, and greatest in the alternating bi-directional type. Attention is called to the fact that of 14 cases, giving no evidence of heart disease, six were of the right ventricular type and only three were of the left type.

TABLE VIII

Average Age and Sex in Relation to Type

	Types				
	Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional	Grand Averages
Average Age	36 years	42 years	50 years	58 years	46 years
					No. of Cases
Males	17 (74%)	18 (81%)	25 (73%)	12 (55%)	68 (73%)
Females	5 (22%)	5 (19%)	9 (24%)	9 (45%)	26 (35%)

(In 2 cases sex was not given.)

The average age incidence increases as the type differs, being least in the right ventricular type and greater in the left ventricular type. This corresponds to expectations in that degenerative changes take place in the left side of the heart before the right as age increases. The average age is greatest in the alternating bi-directional type.

There is a greater percentage of males than females, in all the types considered together. In the alternating bi-directional type there is an approximately equal distribution of the sexes, whereas in the other three types the same relationship obtains as is present in the group as a whole. This seems to be a factor which would tend to cause one to exclude that type from true cases of paroxysmal ventricular tachycardia.

TABLE IX

Related Functional Pathology

	Types			
	Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
Auricular flutter and fibrillation and supraventricular paroxysmal tachycardia	2	0	14	6
Decompensation	5	8	16	13
TOTAL	7 (30%)	8 (38%)	30 (91%)	19 (95%)

It is seen that almost all of the left ventricular and alternating bi-directional types had some functional disturbance whereas only 30 per cent of the right ventricular type showed such disturbance. These figures agree very well with those of Strauss,[49] and further substantiate the evidence that the left ventricular and alternating bi-directional types are associated with more advanced heart disease.

TABLE X

Valvular Disease

		Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
		23 cases	23 cases	35 cases	21 cases
9	Mitral	1 (4%)	2 (9%)	5 (14%)	1 (5%)
10	Aortic	2 (9%)	1 (4%)	3 (9%)	4 (20%)
6	Aortic and mitral	0	1 (4%)	3 (9%)	2 (10%)

It is logical to expect that uncomplicated valvular disease would show a definite relationship to the type of arrhythmia produced. Unfortunately there are only a very few uncomplicated cases of valvular disease in this group. The one case (No. 59) of mitral disease which showed the right ventricular type also had advanced hypertensive heart disease and coronary occlusion. Apparently these complicating conditions did not influence the type of the arrhythmia. Of the five cases with mitral disease which showed the left ventricular type, four (Nos. 13, 36, 52, 65) had complicating factors, such as hypertension, syphilis, coronary disease, exertion, and terminal effects which overcame the influence of the mitral disease and caused the left ventricular type of the arrhythmia. The fifth case (No. 53) had no complicating factors which can account for the left ventricular type.

In the two cases of aortic disease which showed the right ventricular type, no explanation is apparent for the discrepancy. Of the three cases of aortic disease, which showed the left ventricular type, two (Nos. 14 and 79) had complicating factors which might have accounted for the type, but one (No. 25) did not.

Of the three cases with combined aortic and mitral disease, two (Nos. 80 and 69) had no complications which might influence the type, while the other (No. 91) did. Since the complications present in this case would tend to produce the left ventricular type, it may be that which accounts for this instance acting as expected.

In summary it might be said that the influence of these kinds of valvular disease on the type of the accompanying arrhythmia, cannot be determined because of the paucity of uncomplicated cases. Of the 19 cases showing mitral or aortic disease only four were uncomplicated, which number is obviously too small to consider. It must be mentioned, however, that in these four instances there were three which showed the opposite type to that which was expected, while only one (No. 25) was associated with the expected type.

Our conclusions in regard to the influence of valvular disease on this type of arrhythmia are that the strain imparted to the ventricle does not damage that ventricle sufficiently to set up the mechanism involved in this arrhythmia. Instead, it is merely an added factor when some other disease involves the same ventricle, or a less important factor when more serious disease involves the opposite ventricle.

The available autopsy material shows no case of the right ventricular type with marked disease of the right ventricle. One case (No. 48) showed focal myocarditis worse in the left ventricle, and one case (No. 78) with infarction of the posterior wall of the left ventricle.

All three cases of the idio-ventricular type showed infarction, No. 3 in the septum, No. 72 in the posterior and lateral walls of the left ventricle, and No. 85 had a healed infarct in the tip of the left ventricle. Its size is not given.

Eight cases of the left ventricular type were autopsied. Two cases (Nos. 16 and 79) with possibly a third (No. 47) had thrombosis, necrosis, and ecchymotic spots along the left descending coronary artery, respectively, which altered the anterior wall of the left ventricle. Case No. 47 is not definite in this regard. Two cases (Nos. 60 and 77) had definite involvement of the apex; while three cases (Nos. 13, 69 and 82) showed no definite involvement of either ventricle. Four of the eight cases, therefore, showed definite pathology in the left ventricle which might have been a factor in determining the left ventricular type of this arrhythmia in these instances. It should be noted that all of these instances involved the apex or anterior wall of the left ventricle.

Of 11 autopsies in cases of the alternating bi-directional type, three showed thrombosis (Nos. 66 and 72) or infarction (No. 85). Two of

AUTOPSY FINDINGS

Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
Ref. 23; Case 38. Heart weight 320 grams. Cardiac hypertrophy and dilatation. Moderate sclerosis of the aorta and coronaries. Aortic cusps thickened. Right auricle dilated.	*Ref. 3;* Case 3. Most marked changes in the interventricular septum. Dilatation of right auricle and ventricle. Thickening of aortic and mitral valves.	*Ref. 10;* Case 16. Thrombosis of the anterior descending branch of the left coronary artery. Thickening of the walls of the circumflex branch of the left coronary.	*Ref. 18;* Case 28. Cardiac hypertrophy. Syphilitic aortitis. Aortic aneurysm. No coronary thrombosis.
Ref. 29; Case 48. Focal myocarditis (worse in left ventricle and both auricles). Early coronary endarteritis. Acute and chronic myocarditis.	*Ref. 43;* Case 72. (Later changed to Alt. Bi-D. type.) Heart weight 545 grams. Circumflex branch of the left coronary artery showed a narrow lumen. The descending branch of the left coronary was markedly narrow and obliterated by a fresh thrombus. Necrosis of the lower half of the posterior and lateral walls of the left ventricle.	*Ref. 28;* Case 47. (Later changed to Alt. Bi-D. type.) Heart weight 600 grams. Ecchymotic spots along the left descending coronary. Sub-epicardial hemorrhage. Generalized arteriosclerosis. Cardiac hypertrophy and dilatation.	*Ref. 18;* Case 29. Mitral endocarditis. Hypertrophy of right ventricle. Hypertrophy and dilatation of the right auricle. No coronary thrombosis.
Ref. 45; Case 78. Occlusion of the circumflex branch of the left coronary with infarction of the posterior wall of the left ventricle.	*Ref. 50;* Case 85. (Later changed to Alt. Bi-D. type.) Heart weight 950 grams. Healed infarct at tip of left ventricle.	*Ref. 36;* Case 60. Fibrous myocarditis and endocarditis. Thrombosis of a branch of the left descending coronary artery. Coronary sclerosis. Aneurysm at apex of left ventricle.	*Ref. 31;* Case 50. Coronary sclerosis. Many ischemic myocardial lesions.
		Ref. 41; Case 69. (Later changed to Alt. Bi-D. type.) Heart weight 630 grams. Widespread acute inflammatory process in and about walls of smaller coronary arteries.	*Ref. 37;* Case 61. Heart weight 520 grams. General arteriosclerosis. Lumen of the anterior descending branch of the left coronary, two-thirds occluded.
			Ref. 41; Case 66. Heart weight 850 grams. Healed mitral endocarditis. Healed and calcified aortic endocarditis with advanced stenosis. Diffuse fibrosis of the myocardium. Organizing mural thrombi in the ventricles.

AUTOPSY FINDINGS (*Continued*)

Right Ventricular	Idio-Ventricular	Left Ventricular	Alternating Bi-Directional
		Ref. 45; Case 77. Thrombosis of left coronary artery with a large infarct involving the tip of the left ventricle which had ruptured.	*Ref. 41;* Case 66. Organizing pericarditis. Cardiac hypertrophy and dilatation. Anasarca. Pulmonary infarcts.
		Ref. 46; Case 79. Heart weight 400 grams. Syphilitic aortitis with insufficiency. Partial occlusion of left coronary. Myocardial necrosis of left ventricle.	*Ref. 41;* Case 67. Heart weight 659 grams. Left bundle branch showed degenerative changes.
		Ref. 48; Case 82. Megacolon. Heart slightly enlarged. Valves normal.	*Ref. 49;* Case 83. Dilated aorta with two dilatations. Adherent pericardium. Normal myocardium, endocardium and coronary arteries.
		Ref. 8; Case 13. Heart weight 740 grams. Slight coronary sclerosis.	*Ref. 28;* Case 47. See under left ventricular type. No infarction.
			Ref. 41; Case 69. See under left ventricular type. No infarction.
			Ref. 43; Case 72. Thrombosis present. See under idio-ventricular type.
			Ref. 50; Case 85. Infarction present. See under idio-ventricular type.

these cases had a preceding idio-ventricular type of paroxysmal ventricular tachycardia.

In summary we find that there seems to be easily demonstrable autopsy evidence, showing disease of the left ventricle which could account for the left ventricular type of this arrhythmia. This was certain in four out of eight autopsies. One out of three cases gave understandable evidence of disease in the septum which could account for the idio-ventricular type. The evidence from autopsies on cases of the right ventricular type is contradictory, indicating in two out of three cases more disease in the left ventricle. We would emphasize the fact that one of these two cases had infarction of the posterior wall of the left ventricle, in contradistinction to the tendency for involvement of the apex or anterior wall of the left ventricle in the left ventricular type. The low incidence of infarction, three out of 11 autopsies, in the alternating bi-directional type is also emphasized at this time.

The Relationship of Etiology to the Mechanism of the Various Types of This Arrhythmia

The mechanism involved in the production of this arrhythmia has been explained many times. Little, however, has been written which is based upon such a study as this or which takes into account the altered concept of the relationship between ventricular activity and the electrocardiogram which has resulted from the work of Barker, Macleod, and Alexander.[85] The simple statement, that in most of the cases herein studied there was agreement between the type of the arrhythmia and the site of action of the offending heart disease, by no means ends the discussion about paroxysmal ventricular tachycardia. The problem regarding the site of origin of the arrhythmia still remains unsolved. The evidence here presented gives, however, a firm basis for saying that in most instances the site of the new pacemaker in this arrhythmia is in the left ventricle in those cases considered to be of the left ventricular type, according to classical terminology. As previously stated, this need not be considered contradictory to the conclusions of Wilson with reference to bundle branch block.

Primarily we should like, for several reasons, to remove the alternating bi-directional type from the category under discussion. First of all, because the QRS complexes in this arrhythmia are not in every case prolonged as they are in the true ventricular tachycardias where a duration of nearly 0.16 second is almost the rule. Secondly, because while the age incidence and the death rate are highest in this group, marked destruction of the integrity of the ventricular musculature is absent in 75 per cent of the cases and this would suggest that the Purkinje system is more implicated than is the ventricular musculature. Furthermore, this type of the arrhythmia is attributed to excess digitalis administration in nearly 100 per cent of the published cases. This tends to indicate that digitalis is probably solely responsible for the condition since the degree of myocardial damage found in the autopsied cases of this type did not seem sufficient alone to have caused

the arrhythmia, and in the autopsied cases of the other types of this arrhythmia when no greater degree of myocardial damage was found there were likewise grounds to attribute the condition to excess digitalis. Its occurrence in females in the same proportion as in males may also be evidence that the myocardium itself is not the site of the disturbance, because the male is known to be more active than the female and there would be a preponderance of cases among the males, in accordance with the incidence among the other types, if the ventricular musculature were at fault.

With regard to the mechanism of the alternating bi-directional type, we agree, and have witnessed in one unreported case, that it is induced by digitalis administration in the presence of a greatly enlarged heart. We cannot agree to the theory of an alternating block of a bundle branch because the QRS complexes are within the conventional 0.1 second duration. Frequently alternating QRS complexes are of longer duration than others, and this forms the basis for our conception of the arrhythmia. We believe that it is likely to be due to alternating incomplete block of one bundle branch and that it is probably very similar to, if not identical with, the mechanism of coupled beats so often seen in connection with excess digitalis administration. The frequent occurrence under digitalis administration of sinus node, A–V node and His bundle block, complete or incomplete, is ample evidence of the predilection of digitalis for blocking parts of the Purkinje system, and these conditions frequently precede or accompany this type of arrhythmia.

We cannot deny the association of disease of the left ventricle with the left ventricular (classical) type of this arrhythmia. This conforms to the reasonable assumption that the diseased left ventricle would be more likely to set up the arrhythmia than the less diseased right ventricle. We believe that the mechanism is probably due to an irritative lesion of some division of the left branch of the His bundle as suggested by Froment,[86] and probably as a result of extension from the adjacent ventricular musculature.

Likewise it cannot be denied that the right ventricular (classical) type showed a distinct lack of association with those diseases which have been shown to be present in connection with the left type. There was a slight tendency to an association with pulmonary diseases in some instances, especially with pneumonia, and this must be considered. The outstanding features of this type are the lower age incidence, the presence in most cases of only mild heart disease or none, and the fact that this type has the lowest death rate.

It must be said, therefore, that the etiology for the right ventricular (classical) type is not clear. Further evidence must be produced, and more cases must be studied in this manner, before such associations can be made.

Theorizing with regard to this type is irresistible. In the one case (No. 78) in which infarction was found it involved the posterior basal wall of the left ventricle; while in the left type, the four cases of infarction all involved the apex or anterior wall of the left ventricle. We cannot refrain

from pointing out a possible connection between arrhythmias of the right ventricular type having QRS_1 inverted and T_1 upright and QRS_3 upright and T_3 inverted, and cases of coronary occlusion of the posterior basal portion of the left ventricle which have T_{2-3} inverted as observed by Barnes and Whitten.[77] It may be that, when this region is infarcted and the Purkinje system is irritated sufficiently to set up this arrhythmia, the T-waves take direction as reported and the QRS complexes are oppositely directed which would give us our picture of paroxysmal tachycardia of right ventricular origin (classical).

With regard to the influence of a recent tendency to revise our terminology, especially in connection with ventricular extrasystoles [83] and bundle branch block,[87] we would say that at this time our observations with regard to the left ventricular type (classical) do not support such a revision as applied to paroxysmal ventricular tachycardia.

However, we would point out that such observations as were made by Barker, Macleod, and Alexander,[83] Marvin and Oughterson,[84] and Lundy and Bacon,[85] are not yet complete. Neither have they been confirmed in every detail. Until this is done we shall have to hold in abeyance our judgment in this matter. We would emphasize, however, that it seems to be destined that we will have to revise the classical nomenclature of paroxysmal ventricular tachycardia, because proof is gradually accumulating that ventricular extrasystoles, from either right or left ventricle, have different configurations according to the site of origin within the respective ventricles.

Summary

1. We agree that the basic mechanism underlying this arrhythmia is laid down by disease of the ventricular musculature.

2. Considerable evidence is advanced which tends to show a relationship of different kinds of heart disease to different types of this arrhythmia.

3. The mechanism involved in determining the type of this arrhythmia is considered to be heart disease which involves one ventricle more than the other, or a region of one ventricle more than the remainder of the same ventricle, and thereby influences the type according to the ventricle, or its part which has the greater involvement. The suggestion is made that the two types (right and left) may arise from the same ventricle (the left) in different locations. Changed cardiodynamic balance between the two ventricles may be a factor in determining the type by exerting more wear on one ventricle.

4. Basic causes of this arrhythmia are listed in table 6.

5. Basic causes of the arrhythmia are related to the types as follows:

a. Arteriosclerotic, hypertensive, and syphilitic heart diseases are associated in the highest percentage of instances with the left ventricular type.

b. In coronary thrombosis the type is influenced by the location of the infarct.

c. Pulmonary disease, especially pneumonia, was of higher incidence in the right ventricular type.

d. The effect of valvular disease could not be determined.

e. Age exerted an influence as described.

f. The stage of heart disease was associated in a definite manner.

g. Males develop this arrhythmia twice as often as females.

h. Undetermined heart disease was found in 32 per cent of the cases and had a greater relationship to the right ventricular type.

i. No demonstrable heart disease was present in 14 per cent of the cases, most commonly in the right ventricular type.

6. The exciting causes of this arrhythmia are listed in table 5.

7. Exciting causes of the arrhythmia are related to the types as follows:

a. Excess digitalis administration was associated with 100 per cent of the alternating bi-directional type. It was associated with the left type twice as often as with the right.

b. Coronary thrombosis influenced the type according to its location as described.

c. Exertion was most often associated with the left ventricular type.

d. Decompensation and auricular fibrillation and flutter were associated most often with the left type.

e. Nervous and emotional factors are more common in the right ventricular type.

8. Paroxysmal ventricular tachycardia is seen most frequently in the fourth and fifth decades. The youngest patient was aged 16.

9. The arrhythmia is more common in males (73 per cent) than in females (35 per cent).

10. The prognosis is utterly grave in the alternating bi-directional type and only relatively less so in the left ventricular, idio-ventricular, and right ventricular in the order named. In the absence of demonstrable heart disease the prognosis is best in the right ventricular type. Death occurred during the period of observation in 47 cases (47 per cent of the 96 cases whose histories were studied).

11. Reasons are given for not completely ignoring the application of the newer terminology to this arrhythmia.

12. Reasons are given for excluding the alternating bi-directional type, from true cases of paroxysmal ventricular tachycardia. The mechanism of this type is discussed.

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FATAL TULAREMIA

REVIEW OF AUTOPSIED CASES WITH REPORT OF A FATAL CASE *

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The recognition of tularemia as a disease entity is relatively recent. The discovery of the causative organism in 1911 by McCoy, and the studies made by Francis and others, have served to establish the disease on a firm scientific basis. Three hundred and twenty-three case reports were studied by Francis [1] in 1927 and the four clinical types of the disease noted. The mode of infection, clinical signs and symptoms, and mortality rate were well understood at that time. A study of the human pathology of the disease, however, is somewhat more recent. From 1924, when the first autopsied case was reported by Verbrycke,[2] to the present, there has been only a limited amount of human autopsy material available. Verbrycke [2] and Francis and Callender [3] first described the microscopic changes in the lesions in man. Goodpasture and House [4] reported the histopathologic changes in the primary lesion. Permar and Maclachlan,[5] Blackford,[6] Foulger [7] and others have given an accurate description of the pathological changes in the lungs. Foulger, Glazer and Foshay [7] described for the first time lesions on the peritoneum. More recently, meningeal and cerebral lesions due to *B. tularense* have been described by Bryant and Hirsch,[8] and Hartman.[9] While it is probable that the last word has not yet been said as regards the pathology of tularemia in man, it is thought advisable to bring the collected data from these autopsied cases together for study, and at the same time to report a typical fatal case with autopsy findings.

Case Report

S. W., a white male, aged 53, was admitted to the University Hospital on November 17, 1932, complaining of fever, cough, and shortness of breath. He had lived at Mount Pleasant Beach near Glenburnie, Maryland, and had recently been unemployed. On admission, the patient was very ill and toxic, so that no adequate history could be obtained. Later, it was learned that he had handled a dead rabbit brought in by his dog during the last week in October. On November 2 his present illness began with malaise, fever, and chilly sensations, although there were no definite chills. On November 4 a doctor was called who made a diagnosis of "grippe." The patient improved somewhat and was out of bed for a while on November 7. Later that day he became worse, and the following day his physician told him he had pneumonia. He became steadily worse and entered the hospital on November 17 with a tentative diagnosis of pneumonia.

Physical Examination (November 17): The patient was a well developed, fairly well nourished, middle-aged man, obviously desperately ill. He was propped up in

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bed, very dyspneic, and quite cyanotic. The face was flushed and the patient appeared toxic. He had a tight, non-productive cough. Temperature 104°; pulse 140; respirations 50. There was a moderate conjunctivitis. The teeth were dirty and neglected, the tongue dry, the throat moderately injected. There was no cervical rigidity. *Chest:* The heart was negative except for a tachycardia and an occasional extrasystole. Examination of the lungs showed impairment of the percussion note at both bases and many bubbling musical râles, particularly over the areas of impairment. There was no definite change in the breath sounds. *Abdomen:* Distended and tympanitic. *Extremities:* Old amputation of the right lower leg. Over the dorsal surface of the right hand between the index and third fingers was a freshly healing ulcer about the size of a 25 cent piece. There was a similar lesion on the left hand near the proximal joint of the thumb. The epitrochlear and axillary lymph nodes were not palpable.

Laboratory Findings: The urine contained large quantities of albumin; occasional white blood cells and red blood cells, and a few granular casts were noted. The blood picture was normal except for a leukocytic count of 4,000 on November 17 with 78 per cent polymorphonuclear leukocytes in the differential smear. On November 18, the leukocytes had fallen to 3,750. The Kolmer was negative. Because of the history of handling a dead rabbit and the ulcers on the patient's hands, an agglutination for tularemia was requested. This was reported by the University Hospital Laboratory on November 17 as complete at 1:200 and partial at 1:800. A second specimen sent to the Baltimore City Health Department on November 18 agglutinated *B. tularense* completely at 1:320. A blood culture yielded after 72 hours an organism morphologically *B. tularense*. This organism produced lesions typical of visceral tularemia in liver, spleen and lymph nodes when inoculated into a guinea pig.

The patient was transferred to the oxygen chamber soon after admission and lapsed into unconsciousness that afternoon. The following day there were palpable glands in the right axilla, increase in abdominal distention with some tenderness, and pulmonary edema. The temperature rose to 106° and the patient died on November 18, one day after admission and 17 days after the onset of his illness. Permission for partial autopsy was granted.

Postmortem Examination: On external examination the following pathological changes were observed. Two ulcers were noted on the hands. One was situated on the dorsal surface of the right hand and was irregularly circular. It measured 2 by 1.5 cm. The edges were raised, discolored, and firm, and the base was rather punched out and covered with a dry crust. A similar lesion was present on the left thumb near the metacarpo-phalangeal articulation. There was no edema, little inflammatory reaction, and no evidence of lymphangitis in either of these regions. A shotty lymph node was present near the right elbow. A moderately soft gland was present in the right axilla. This node measured about 4 cm. in diameter. The upper chest and neck were markedly suffused and livid. A healed old amputation was noted at the middle third of the right tibia with some muscular atrophy of the thigh on that side.

On opening the thorax a fibrinous exudate was found in both pleural cavities, together with about 100 c.c. of cloudy fluid in each side. Patchy organization and friable adhesions were present between the parietal and visceral pleurae. The right lung was extremely heavy and voluminous, and the fibrinous pleural exudate was most marked on the lateral and posterior surfaces. Discrete caseous foci could also be seen in the subpleural tissue. The upper two lobes were almost confluently consolidated and multiple nodular foci could be palpated in the lower lobe. Crepitation was almost entirely absent in this lung. On the sectioned surface a greyish, granular exudate was observed and in the lung tissue discrete areas of caseous necrosis or abscess formation were present. (Figure 1.) A thick, blood tinged exudate could be expressed from the parenchyma in areas, and the larger bronchi contained a similar material.

The left lung was very similar to the right, with a fibrinous pleuritis more limited to the apical region, and a pneumonic consolidation involving for the most part the upper lobe. The same discrete foci interspersed with a granular and partly necrotic exudate were observed on the cut surface. The hylic and mediastinal nodes were swollen, and multiple greyish white foci of necrosis were present.

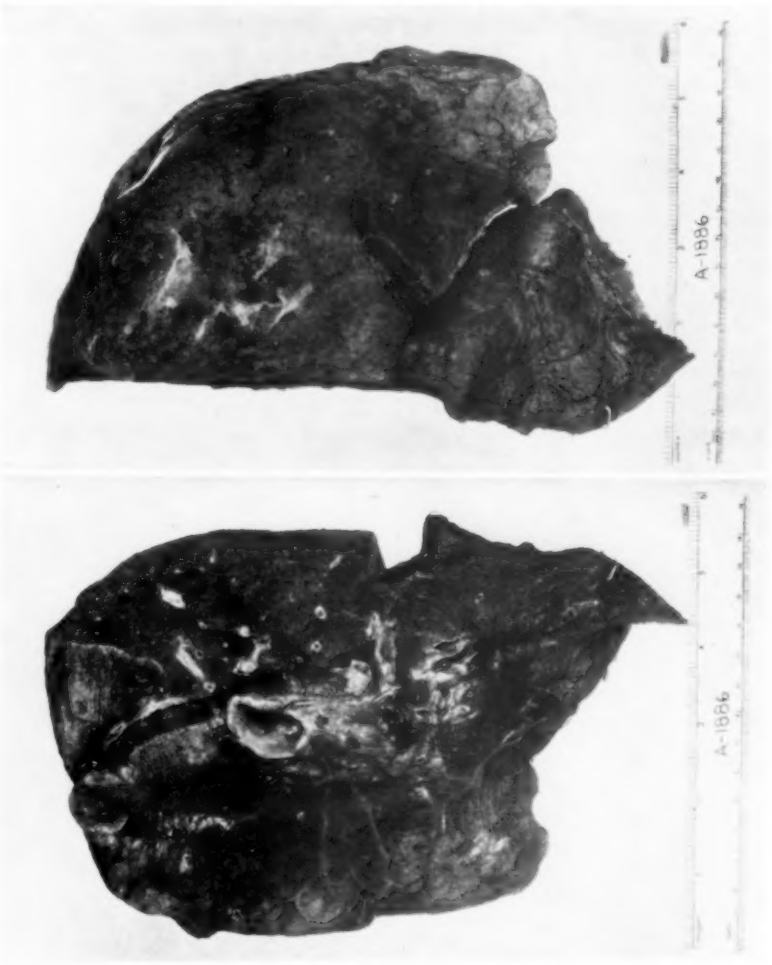

FIG. 1. Right lung, showing medial cut surface and lateral pleural surface. Note consolidation of upper lobe with foci of necrosis. The hylic nodes also show necrosis.

The heart weighed 375 grams. The myocardium of the left ventricle measured 18 mm. in thickness and grossly was somewhat swollen and pale. The heart was not otherwise unusual. The aorta showed early atherosclerosis. The liver was enlarged, soft and pale, and weighed 2,200 grams. Several pale, white, nodular foci were observed under the capsule. On the cut surface these multiple foci of necrosis were more evident. On the capsule of the spleen and on the peritoneal surface of the diaphragm in this vicinity, there was noted a fibrinous exudate. (Figure 2.) A few

coils of distended intestine were slightly adherent to the spleen and diaphragm. The spleen weighed 350 grams. Multiple yellowish foci of necrosis were noted under the capsule. The pulp was reddish grey, semifluid, and tended to overflow the capsule. The Malpighian bodies were practically obliterated by this swelling of the pulp. Yellowish areas of necrosis were present, ranging from miliary size to 4 to 6 mm. The kidneys, aside from some enlargement and slight subcapsular edema, appeared normal. They weighed 250 grams each. A moderate amount of hypertrophy of the lateral lobes of the prostate was present without urinary retention.

FIG. 2. External and cut surface of spleen showing fibrinous exudate on the capsule and foci of necrosis in the pulp.

The bladder appeared normal. The pancreas and adrenals were not unusual. The gastrointestinal tract showed no gross lesions.

The brain was not removed.

Microscopic Examination: The primary lesion on the hand was not studied histologically.

Sections from the axillary node showed both discrete foci and confluent areas of necrosis. These necrotic areas were rather acellular, being for the most part composed of granular débris and fragmented nuclear particles. About the margins of these areas were many large, fat-laden phagocytic cells and lymphocytes. Little or no productive reaction was observed. No giant cells were seen. The general architecture of the node was not destroyed, although some edema was noted.

The lesions in the liver appeared miliary in size, a few being slightly larger than a liver lobule. These foci of necrosis in the liver were for the most part situated in portal vein zones. A rather sharp line of demarcation was present between the necrotic material and the liver substance. These foci appeared as areas of acute coagulation necrosis, being composed of granular débris, degenerated liver cells, with pycnotic nuclei and fragmented nuclear particles. Cellular infiltration of a monocytic character with a few polymorphonuclear leukocytes was present about the periphery.

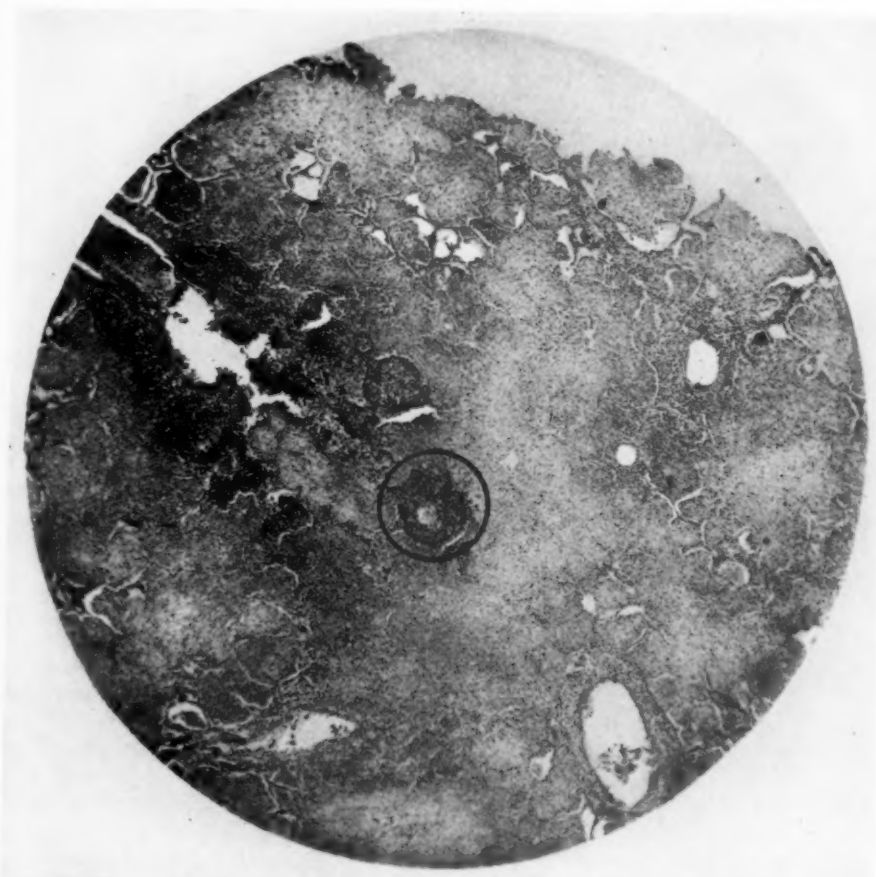

FIG. 3. Areas of necrosis in consolidated lung. × 35.

A few poorly formed giant cells were observed. Some fatty changes were present in the parenchyma but were not related to the foci of necrosis.

Sections from the lung showed an exudative pneumonia that confluently involved large areas, in addition to localized foci of necrosis. Many areas showed no air containing alveoli. This exudative process was widespread in sections from the upper lobes. All constituents of the exudate: lymphocytes, polymorphonuclear cells, fibrin, and red blood cells, were in a poor state of preservation, and in localized areas there was necrosis of this exudate as well as lung tissue. (Figure 3.) These discrete areas appeared identical with the lesions in the liver, node and spleen. A few of these foci were in relation to bronchi and in these areas the bronchial lumen was filled with débris and coagulated exudate. Although the process in many ways presented a similarity to caseous tuberculous pneumonia, no cavitation or acinar-like lesions were seen, and no giant cells observed. One of the most outstanding features was the marked interstitial involvement. There was edema and lymphocytic infiltration of the

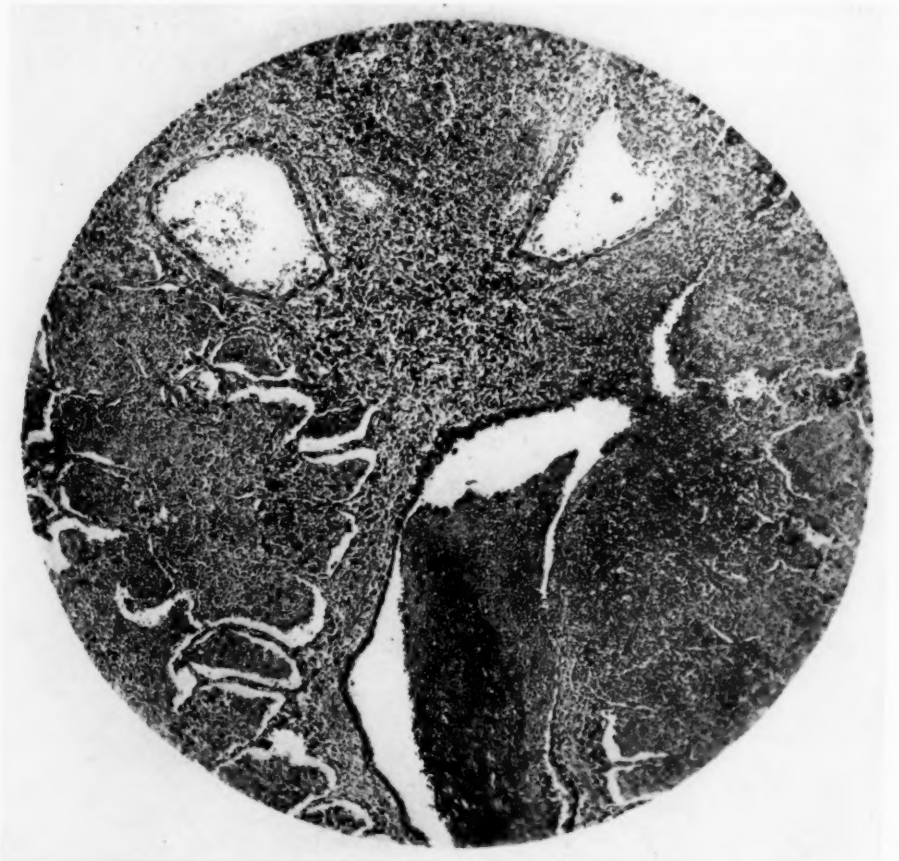

FIG. 4. Interstitial edema and lymphocytic infiltration in the peribronchial tissue with an area of necrosis ulcerating into a bronchus. × 120.

perivascular and peribronchial tissue. (Figure 4.) The walls of the smaller vessels seemed to be participating in this inflammatory process. There was swelling of the subendothelial connective tissue of many of the small arterioles and venules, in some

instances resulting in thrombosis. This condition may have contributed to the production of the necrosis. (Figures 5 and 6.)

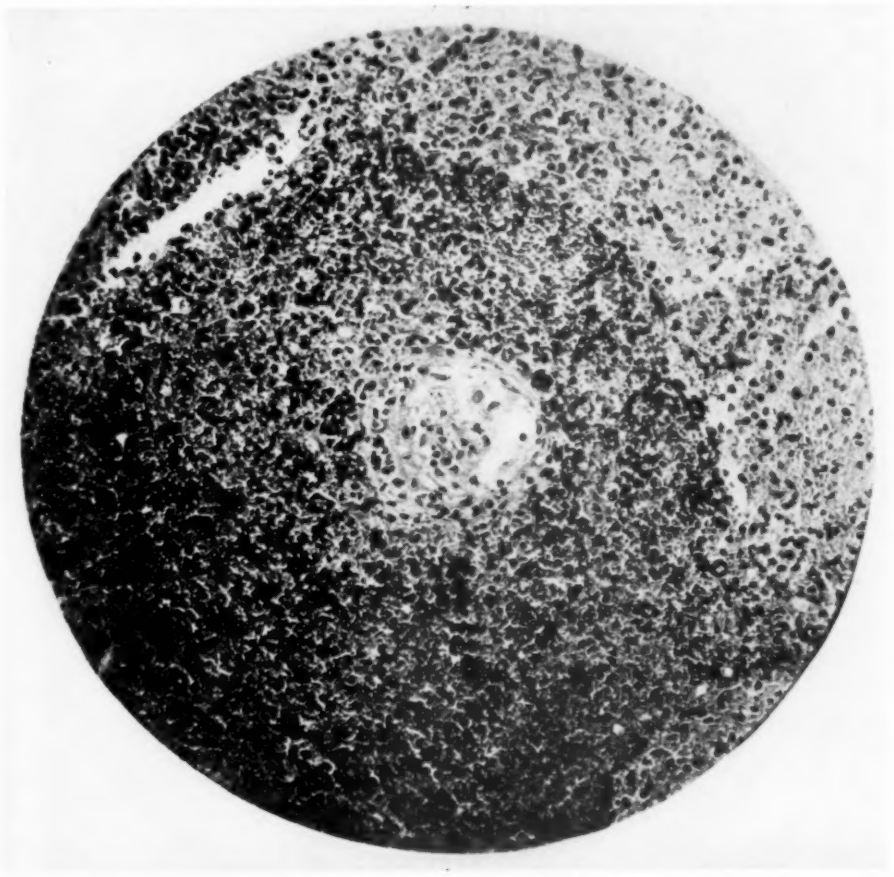

FIG. 5. High power of circular area from figure 3, showing thrombus in vessel surrounded by a mantle of necrosis. × 300.

Sections from the spleen showed a normal appearing pulp and Malpighian body element with typical areas of acute necrosis identical with those described in the liver and node.

A section from the diaphragm presented a generalized diffuse reaction consisting chiefly of lymphocytes lying beneath a layer of degenerated and hyalinized fibrin. No local necrotic areas were noted in this exudate.

Some tubular degeneration was noted in the sections from kidneys but there were no changes suggesting specific pathology. The histologic changes observed in the heart, pancreas, and adrenals were not unusual. All human tissue in which histological tularemic lesions were observed, were stained by the method employed by Foshay [10] to demonstrate the organism in the lungs. The marked nuclear fragmentation and the accumulation of chromatin particles in and around these lesions made the demonstration of any organism uncertain. Tubercle bacilli were not found.

Bacteriological Examination: At autopsy, material from the necrotic foci in the

spleen was macerated and inoculated subcutaneously into a guinea pig. This animal succumbed in five days with the typical visceral lesions of guinea pig tularemia. Another guinea pig was injected with 1 c.c. of a broth culture which had been inoculated with blood taken from the heart of the patient at autopsy. This culture was injected intraperitoneally but this animal developed no tularemic manifestations.

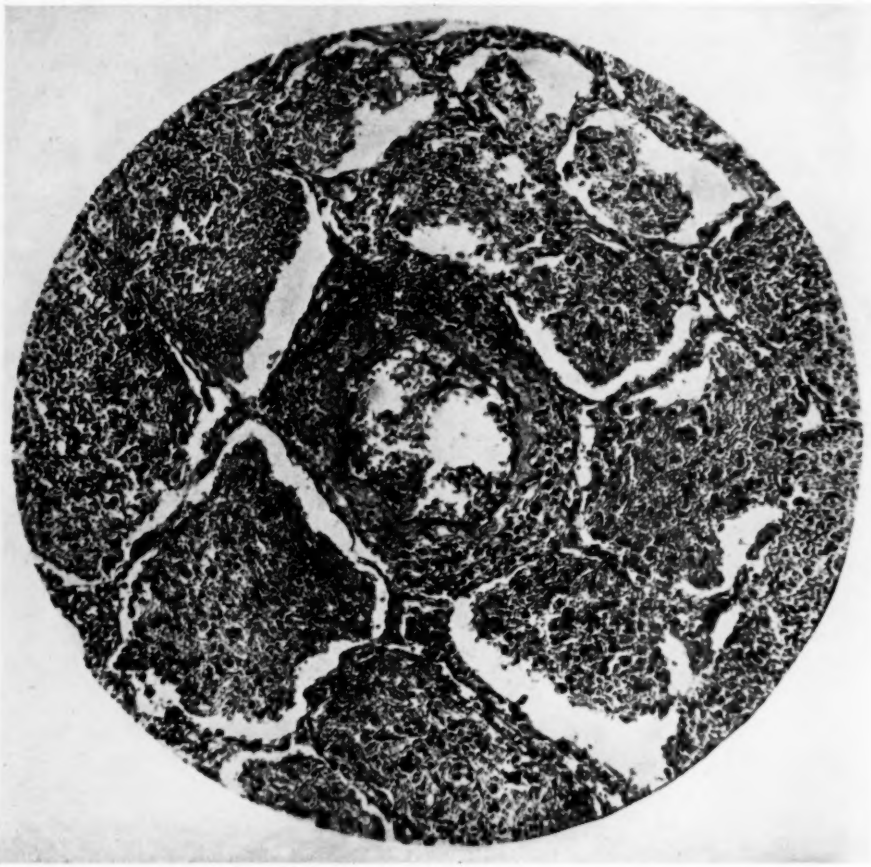

FIG. 6. Vessel in pneumonic area in lung showing subintimal edema. × 230.

Blood from the patient taken the day before death was cultured on veal brain broth and dextrose broth. The veal brain broth contained calcium carbonate to produce semi-anaerobic conditions. A growth was obtained in both; it was more profuse in the veal brain broth. This culture contained an organism morphologically identical with *B. tularense*. A guinea pig was inoculated intraperitoneally with two cubic centimeters of the veal brain broth culture. This animal died in six days and at autopsy typical tularemic lesions were found.

A search of the literature disclosed reports of 14 fatal cases of tularemia with autopsy. These cases together with our own have been summarized as to clinical and pathological findings in tables 1 and 2. In discussing these tables, the first fact worthy of note is that the 15 autopsied cases re-

ported appear under the names of 15 different authors or groups of authors. That there are so few autopsy reports speaks for the low mortality of tularemia.

The mode of infection was contact with rabbits in 13 cases; contact with an opossum in one; and the bite of a deer-fly in one. The average incubation period in 12 cases (not mentioned in remaining three) was 3.5 days. This exactly corresponds with the incubation period as given by Francis,[1] although one might expect to find a shorter than average incubation period in these fatal cases. The average duration to death was 20 days; Blackford's[6] case, 41 days, being the longest, and the fulminating case of Simpson,[13] four days, being the shortest. Fourteen cases were of the ulceroglandular type, and one should probably be classed as typhoidal. Clinically, pneumonia or other lung involvement was a very frequent finding: bronchopneumonia was diagnosed in six cases; lobar pneumonia twice; lung abscess once; and pleural effusion once. The lung involvement often furnished the presenting symptoms and very frequently was the cause of death. In five cases, however, the lung findings were negative.

The average leukocyte count in 12 cases was 10,800; the highest 22,600, in the case of Goodpasture and House[4]; the lowest 3,750, in our own. Simpson[17] gives the leukocyte count in tularemia as ten to fifteen thousand. Poor resistance is suggested by the low average of these counts or rather by the many low leukocyte counts shown in the table. Agglutination tests for tularemia were uniformly negative during the first eight days and uniformly positive thereafter, with steadily increasing agglutination titer as the disease progressed. Thus Blackford's[6] case (25th day) agglutinated at 1:5120, Hartman's[9] case (18th day) 1:1280, Bardon and Berdez's[12] (12th day) 1:80. Massee's[16] case demonstrates very well the increasing agglutination titer.

In analyzing the pathological data from the above chart, we find that a lesion at the portal of entry of the organism was found in all but one case, and that the ulceration at this site usually remained until death. The regional glands were involved in all cases; the presence of large caseous peribronchial glands in Permar and Maclachlan's[5] case, which was without external evidence of a portal of entry, lends credibility to the idea that this case possibly represents an instance of primary respiratory tract infection. In 12 of the 15 cases, either gross or microscopic lesions were noted in the liver. Eleven cases showed necrotic foci in the spleen.

The lungs, next to the regional nodes, were the most constant site of involvement. In Francis and Callender's[3] case the postmortem examination was limited to an inspection of the abdominal viscera, but the patient had clinical signs of pneumonic involvement. The remaining 14 cases all showed some pulmonary lesion at autopsy. The most constant lesions in the lungs in these autopsied cases were necrotic foci. This finding was present in 11 cases. Extensive pneumonia was observed eight times; pleural effusion three times, and cavitation and abscess twice.

TABLE I

Clinical Features in Autopsied Cases of Tularemia

Author	Mode of Infection	Incubation	Duration to Death	Type of Infection	Pneumonia or Other Lung Involvement	Leukocyte Count	Agglutination
Verbrycke [2]	Housewife dressing rabbit	5 days	18 days	Ulceroglandular	No signs mentioned	12,000 15,400 19,200	1 : 80 (16th day)
Francis and Callender [3]	Farmer Fly-bite	Unknown	26 days	Ulceroglandular	Right upper; rusty sputum	Not reported	Not reported
Schumacher [11]	Farmer dressing rabbit	Not mentioned	29 days	Ulceroglandular	No clinical signs	Not reported	Negative (8th day) 1 : 1280 (19th day)
Bardon and Berdez [12]	Farmer handling rabbit	1 day	15 days	Ulceroglandular	Bronchopneumonia, both bases	Not reported	1 : 160 (10th day)
Goodpasture and House [4]	Dressing rabbits	1 or 3 days	14 days	Ulceroglandular	No clinical manifestations	15,000 19,000 22,600	Negative (8th day) 1 : 80 (12th day)
Simpson [13]	Market worker handling rabbits	7 days	4 days	Ulceroglandular	Extensive bronchopneumonia	15,400	Negative
Palmer and Hansmann [14]	Housewife handling rabbit	3 days	8 days	Ulceroglandular	No clinical signs	9,000	Negative

Bruecken's case (as reported by Francis) is not included in this table. The patient died of hemolytic streptococcus septicemia 21 weeks after onset of tularemia.

TABLE I (*continued*)

Author	Mode of Infection	Incubation	Duration to Death	Type of Infection	Pneumonia or Other Lung Involvement	Leukocyte Count	Agglutination
Bunker and Smith [15]	Hunting and skinning rabbits	5 days	14 days	Ulceroglandular	Signs of effusion	9,400 6,600	Negative at first; low titer last day
Massee [16]	Marketman dressing rabbits	Not mentioned	18 days	Ulceroglandular	Bronchopneumonia both bases	11,500	Negative (8th day) 1 : 40 (15th day) 1 : 320 (18th day)
Permar and Maclachlan [5]	Dressing rabbits	1 day	17 days	Probably typhoidal	Lobar pneumonia, rt. upper (late)	7,125 5,200 (17th)	Negative (7th day) 1 : 320 (17th day)
Bryant and Hirsch [8]	Chef dressing rabbit	3 days	16 days	Ulceroglandular	No clinical manifestations	7,100	1 : 40 (14th day)
Hartman [9]	Butcher skinning rabbit	4 days	36 days	Ulceroglandular	Many râles, left base	11,500 11,400	1 : 640 (15th day) 1 : 1280 (18th day)
Foulger, Glazer and Foshay [7]	Housewife handling rabbit	4 days	21 days	Ulceroglandular	Bronchopneumonia, right	12,000 4,000	1 : 40 (2nd week)
Blackford [6]	Negro handling opossum	4 days	41 days	Ulceroglandular	Lung abscess, cough, with foul bloody sputum	8,200	1 : 40 (9th day) 1 : 5120 (25th day)
Gundry and Warner	Handling rabbit	3 to 5 days	17 days	Ulceroglandular	Pneumonia, bilateral	4,000 3,750	1 : 200 (16th day) 1 : 320 (17th day)

TABLE II

Pathological Findings in Autopsied Cases of Tularemia

Author	Primary Site	Glands	Liver	Spleen	Lungs	Tularemic Lesions Elsewhere	Positive Animal Inoculation
Verbrycke [2]	Right index finger	Regional, enlarged	Studded with necrotic areas	Multiple necrotic foci	Caseous peribronchial nodes; acute purulent bronchopneu., 100 c.c. in each pleural cavity		Guinea pigs, mice, rabbits, inoc. with spleen
Francis and Callender [3]	Fly bite on rt. side neck	Post auricular, right	Incomplete autopsy: abdomen exposed. Spleen found studded with necrotic foci.				From blood fourth day. Node and spleen at autopsy
Schumacher [11]	Ulcer, left index finger	Enlarged left axillary	No lesions	Enlarged	No lesions observed	Peritonitis. Ulcers-cecum	Not mentioned
Bardon and Berdez [12]	Left middle finger	Enlarged axillary	Studded with nodular areas of necrosis	Areas of necrosis	Bronchial glands, caseous nodes in pleura; extensive pneumonia, both bases	Nodules, kidneys	None reported
Goodpasture and House [4]	Rt. index finger	Caseous axillary nodes	Enlarged with miliary caseous foci	Enlarged areas of necrosis	Peribronchial nodes, caseous. No lesions, lung. 200 c.c. left pleura	Mediastinal and retroperitoneal nodes	From organism recovered ante-mortem blood. Axillary node at autopsy

TABLE II (*continued*)

Author	Primary Site	Glands	Liver	Spleen	Lungs	Tularemic Lesions Elsewhere	Positive Animal Inoculation
Simpson [13]	Finger	Enlarged and caseous axillary nodes	Multiple necrotic foci	Necrotic foci	Extensive pneumonia with necrosis		
Palmer and Hansmann [14]	Finger	Enlarged and caseous axillary nodes	Areas of necrosis	Yellow necrotic areas	Inconsiderable bronchopneumonia		From heart's blood and axillary tissue
Bunker and Smith [15]	Finger	Caseous regional nodes	No lesions observed	Necrotic foci	28 oz. effusion. 6 to 8 foci of necrosis		From finger, eighth day; sputum twelfth; autopsy fourteenth
Massee [16]	Thumb	Regional nodes, enlarged	No gross lesion but microscopic necrotic foci	No lesions	Pneumonic consolidation, both bases. Confluent lobular pneumonia		From thumb, spleen and lung; all died with typical lesions
Permar and Maclachlan [5]	Not found; possibly resp. tract (?)	Peribronchial nodes enlarged and necrotic	Studded with areas of necrosis	Recent infarct	Lobar consolidation, rt. upper. Focal areas of necrosis		Not reported
Bryant and Hirsch [8]	Rt. middle finger	Caseous axillary and supraclavicular	Miliary necrotic foci	Multiple necrotic nodules	Exudative pneumonia with focal necrosis	Leptomeningitis; necrosis subapp. tissue	Guinea pig inoculation with spleen, liver and lungs; typical lesions

TABLE II (*continued*)

Author	Primary Site	Glands	Liver	Spleen	Lungs	Tularemic Lesions Elsewhere	Positive Animal Inoculation
Hartman [9]	Base of left thumb	Left axillary and peribronchial	Necrotic foci present	Microscopic necrosis	Necrotic nodules, left lung	Necrotic areas brain substance; Encephalitis	None reported
Foulger, Glazer and Foshay [7]	Finger	Axillary nodes	Multiple necrotic areas	Necrotic foci	Caseous abscess, right middle and lower, lobular pneumonia	Peritonitis	Guinea pig inoculated from liver abscesses
Blackford [6]	Left third finger (?)	Enlarged, necrotic left axillary node	No lesions	No lesions	Cavities, rt. lower. Thrombosis, rt. pulmonary art. Necrosis in lung		Not reported
Gundry and Warner	Dorsum of rt. hand and thumb	Epitrochlear and axillary, right	Miliary foci of necrosis	Enlarged necrotic foci	Confluent lobular pneumonia, both uppers. Necrosis Fibrinous pleuritis	Localized peritonitis, diaphragm	From blood culture antemortem. From spleen at autopsy

The occurrence of lesions other than those of the glands, liver, spleen and lung was infrequent. Bardon and Berdez[12] found lesions in the kidneys; Foulger, Glazer and Foshay,[7] Schumacher[11] and ourselves, lesions on the peritoneum. A new field has been opened up by the more recent reports of Bryant and Hirsch,[8] and Hartman,[9] who describe lesions in the brain and meninges.

As regards bacteriology, animal inoculation has proved positive in the majority of these autopsied cases. In two instances the organism was stained in the tissue, Massee,[16] Foulger, et al.[7] In our own case the organism was cultivated directly from the antemortem human blood.

Comment

It is evident from the analysis of the autopsied cases that the pulmonary lesions of tularemia are an important feature of the visceral pathology. This fact has been stressed also by Blackford[6] and by Permar and Maclachlan.[5] Francis[1] in his study of 24 fatal cases of tularemia found that more than one-third had shown clinical evidence of pneumonia. Tureen[18] noted that there was a high mortality rate in those cases in which pneumonia developed.

Various types of pulmonary lesions have been described, bronchopneumonia, lobar pneumonia, cavitation, abscess, and pleurisy, with or without effusion; but it would appear that the most characteristic lesion is a lobular type of pneumonia containing foci of necrosis. In the earlier cases that came to autopsy the localized necrotic lesions only were considered to be due to tularemia. The more diffuse patches of consolidation were interpreted as a secondary bronchopneumonic process. More recently the closer histological study of these pneumonic areas and the demonstration by Massee[16] and by Foulger, Glazer and Foshay[7] of the *B. tularense* in the pulmonary exudate has led to the acceptance of the view that all of the pulmonary pathological lesions are due to the tularemic infection. Our own histological observations are in accord with those of Permar and Maclachlan.[5] We feel that the outstanding features of the pulmonary lesions may be summarized as follows: the process is a confluent lobular pneumonia with marked involvement of the interstitial tissues; the exudate contains a predominant number of lymphocytic cells; vascular lesions are found characterized by subintimal edema, often associated with thrombosis. These vascular occlusions offer the best explanation of the areas of necrosis which are typical features of tularemic pathology.

Attention should be drawn again to the resemblance of pulmonary tularemia to pulmonary tuberculosis. It is probable that at times the gross pathological pictures have been confused. It should be noted also that the clinical picture of tularemic pneumonia might readily be mistaken for an ordinary bronchopneumonia or an atypical pneumococcic lobar pneumonia.

The second most frequent site of involvement in the fatal cases reviewed was the central nervous system. Francis and Callender[3] in 1927,

in discussing a fatal case without autopsy, first suggested on the basis of the clinical findings that meningeal or cerebral lesions were probably present. In 24 fatal cases reported by Francis [1] in 1928, it was noted that five had died in coma. Later Bryant and Hirsch [8] in a case with clinical symptoms of meningitis reported a marked pleocytosis in the spinal fluid and at autopsy the presence of a leptomeningitis. Hartman's [9] case was admitted with fever and delirium; at autopsy a diffuse encephalitis was found.

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FRAGILITAS OSSIUM IN FIVE GENERATIONS*

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We have had the opportunity of seeing eight cases of fragilitas ossium in one family covering three generations, and have been able to trace the disease in five generations. We present a report of three of these cases.

Case I

Dora B., 43, married, was admitted to the Israel Zion Hospital in January 1932 with the complaint of headaches and dizziness of several months' duration.

Previous History. The patient began to walk at the age of five years. In childhood she had abscessed ears which caused some impairment of hearing. Her menstruation had always been irregular. The last period was in October 1931. She had had five children. There had been no miscarriages. As long as she could remember, she had been subject to "sprains" of her hands and feet from the slightest cause, such as lifting a pail. On one occasion she sustained a fracture of her thigh from tripping.

Physical Examination. Short, obese female, weighing 194 pounds. The sclerae were of a striking blue color, a cerulean blue. On inquiry as to the presence of blue sclerotics in any other members of her family, she volunteered the information that her family was known as "the people with the blue eyes." Inspection also revealed deformities of the hands produced by flail-like joints. Many members of her family were double jointed. Her feet were markedly flattened from ligamentous relaxation.

Her blood pressure was 170/110. The eyegrounds showed slight vascular sclerosis. Otherwise physical examination was negative.

Urine: sp. gr. 1020; faint trace of albumin; many calcium oxalate crystals, occasional pus cells, no casts. Red blood cells 4,000,000; white blood cells 10,600; hemoglobin 95 per cent. The differential count showed 43 per cent of polynuclears, 47 per cent lymphocytes, 9 per cent mononuclears, and 1 per cent basophiles.

Blood: Chemical analyses:

Urea N	16. mg.
Uric A	4.2 mg.
Chlorides	475. mg.
Cholesterol	275. mg.
Glucose	75. mg.
Creatinine	1.5 mg.
Phosphorus	3. mg.
Calcium	8.0 mg.

The blood calcium determination was repeated and gave a reading of 9.8 mg.

Extensive roentgen-ray studies of the osseous system were made. Two of these roentgenograms are shown in figures 1 and 2.

A diagnosis of fragilitas ossium and essential hypertension was made.

Case II

A. B., age 9 1/2, daughter of patient in case 1.

Previous History. In infancy the patient was breast fed for five weeks, but the

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mother then sustained a shock and was unable to continue nursing so that artificial feeding was instituted. The child suffered from frequent attacks of vomiting and diarrhea. At the age of five weeks, the mother lifted the child out of the bath tub and fractured an arm. At the age of eight months, a leg was fractured. At 19

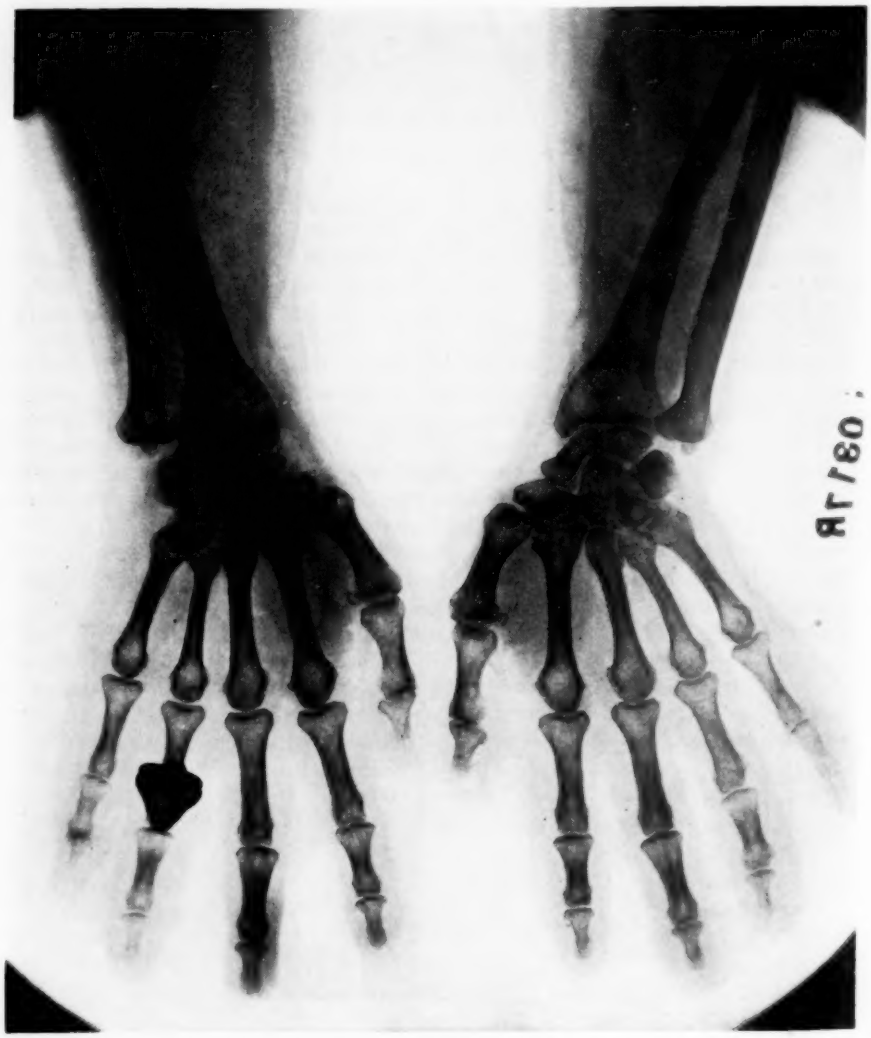

FIG. 1. Phlebosclerosis of both forearms. Extremities of metacarpals broadened, with shaft narrowing, producing dumb-bell effect.

months, the child began to walk. At 23 months, she fell from a chair and fractured her left thigh. At 30 months, she fractured the left humerus, and this was followed in two weeks by a fracture of the other humerus. In all there have been 18 recorded fractures. One year ago, her school physician noticed a painless spinal curvature. Examination at a hospital disclosed a fractured vertebra, for which she is still wearing a spinal brace. Since birth the child's extremities have always been cold. She has been double jointed and she bruises easily.

Physical Examination. Weight 79 pounds. The sclerae are of a deep blue color. There is a marked spinal scoliosis, and a well developed pes planus. There are many ecchymotic spots over the body.

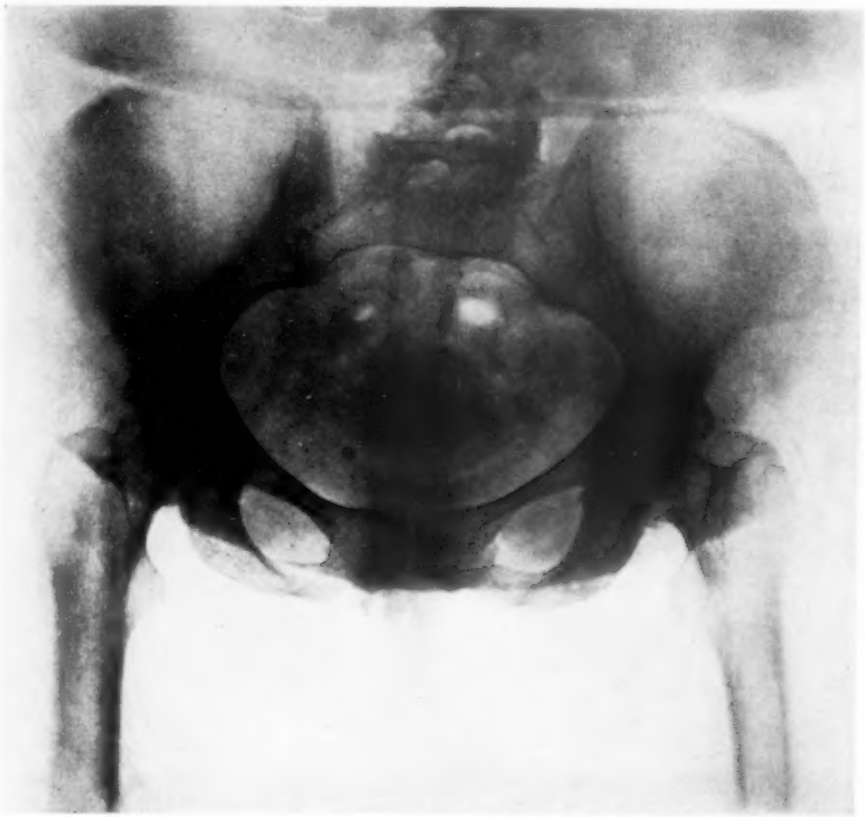

FIG. 2. Flat pelvis. Note compensation of the transverse diameter for the foreshortened antero-posterior. Defect lower left aspect of the body of the third lumbar vertebra, incident to mild trauma.

Blood: Chemical analyses (January 1932):

Calcium	5.0
Phosphorus	3.5
Cholesterol	265.0
Chlorides	475.0

The calcium and phosphorus determinations were repeated about four weeks later and were reported as calcium 9.0 and phosphorus 5.5. The basal metabolic rate was plus two. Numerous roentgenograms of the osseous system were made, two of which are shown in figures 3 and 4.

CASE III

S. R., age 13, a niece of the patient in case 1.

Previous History. The child had been breast fed until 15 months of age. She was then placed on whole milk to the amount of five to six quarts daily, to the ex-

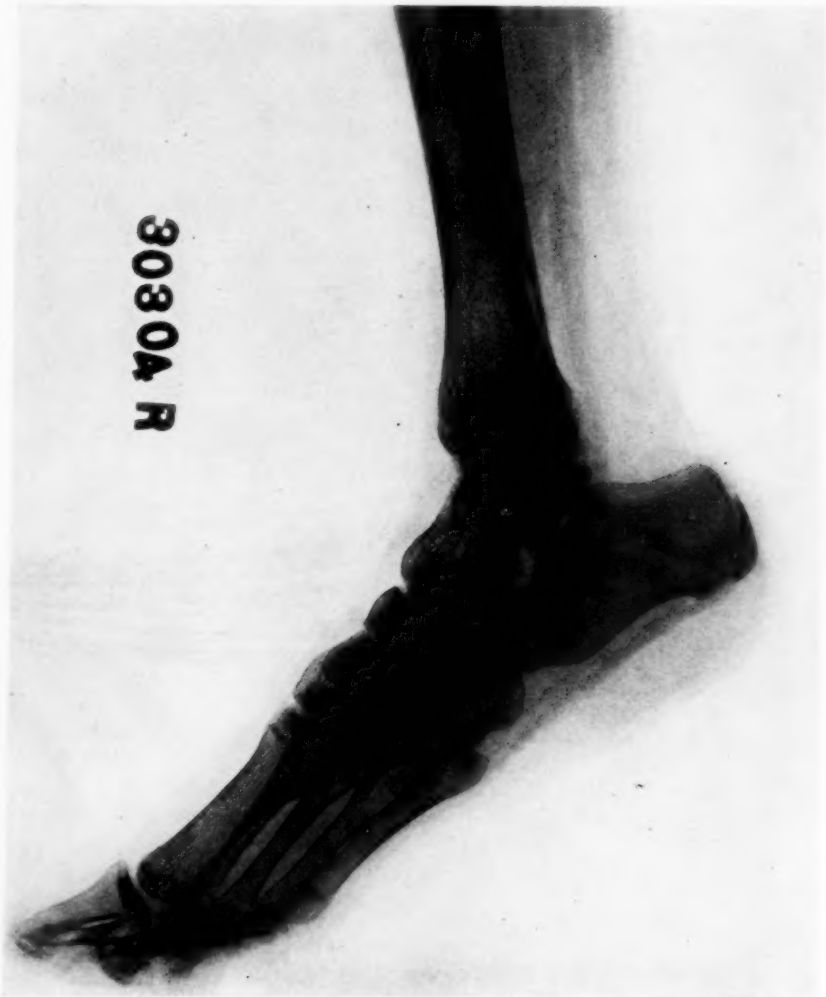

FIG. 3. Characteristic osteoporosis of lower tibia and fibula, tarsal and metatarsal bones. Note thinness of cortex of all bones, with well demarcated trabeculation throughout. Plantar arch flattened, common to ligamentous relaxation.

clusion of other foods. At the age of 18 months, she fractured her right hip, at 22 months the right leg, and two weeks later the right wrist. At the age of four years, the right leg was fractured and two months later the right ankle. Since then she has had many other fractures from the slightest trauma. Since the age of eight months, she has had a discharging left ear. The menses began about five months ago.

Physical Examination. A pituitary type of individual, weighing 131 pounds, with blue sclerae, relaxed joints, marked pes planus and genu valgus. The teeth were deformed and defective.

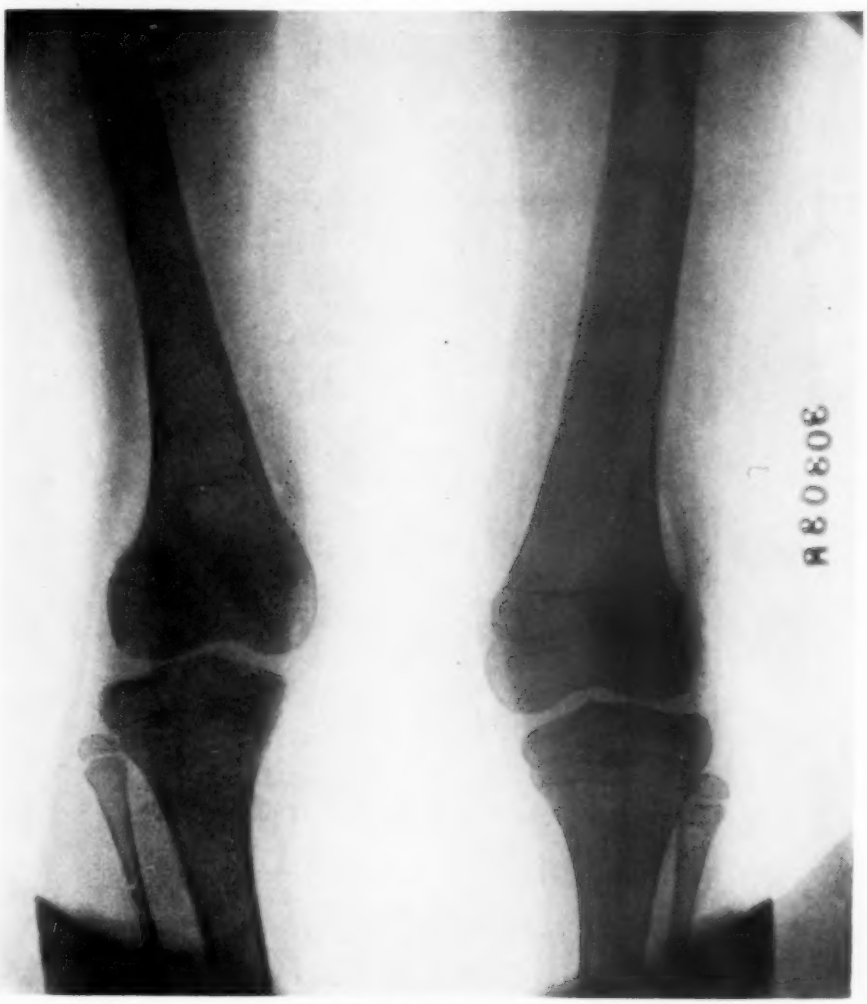

FIG. 4. Subperiosteal proliferation (clinically painless) lower right femur, of recent mild traumatic origin. Note curvature of bone above and slight sclerosis, the end result of previous fractures. Metaphyses and epiphyses of both knees markedly reticulated and decalcified. Dense transverse metaphyseal lines, indicating progressive zones of calcification.

Blood: Chemical analyses:

	January 1932	*March 1932*
Chloride	480	450
Cholesterol	290	
Phosphorus	3.5	5.5
Calcium	7.0	9.7

The basal metabolic rate was plus two. Roentgenographic studies were made; two of these roentgenograms are shown in figures 5 and 6.

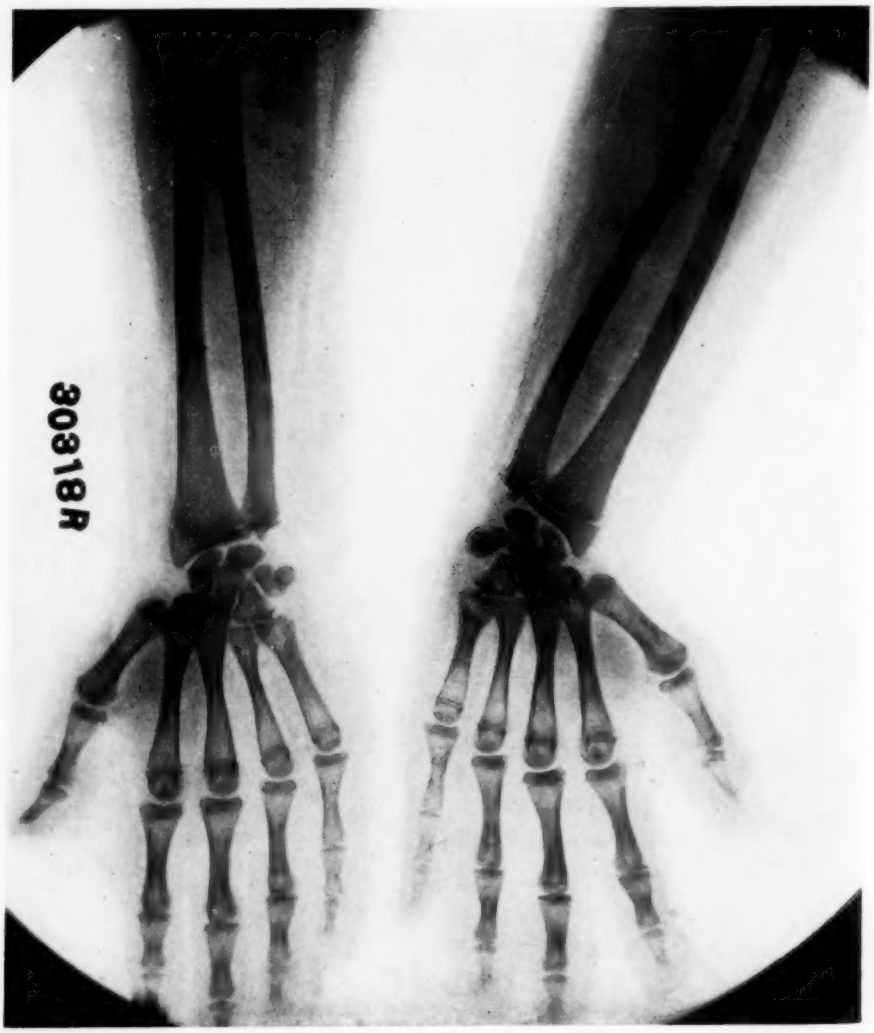

FIG. 5. Pronounced phlebosclerosis. Subcutaneous venous network visible throughout the length of both forearms.

We append an illustration of the family tree of these patients (figure 7). We have personally seen members of three generations of this family; and we have obtained a history of the existence of this disease in two other generations from three independent sources.

DISCUSSION

The three cases studied were 43, 13 and 9 1/2 years of age. Aside from the characteristic findings in the roentgen-ray studies, a distinct prominence

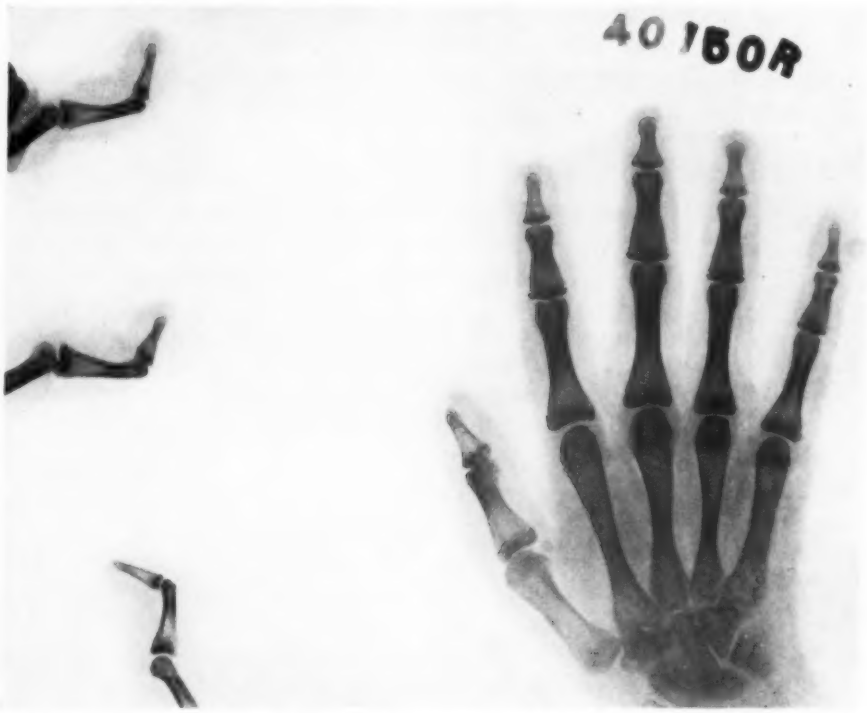

FIG. 6. Osteoporosis, with articular relaxation, giving rise to double-jointed index finger. Dumb-bell metacarpals.

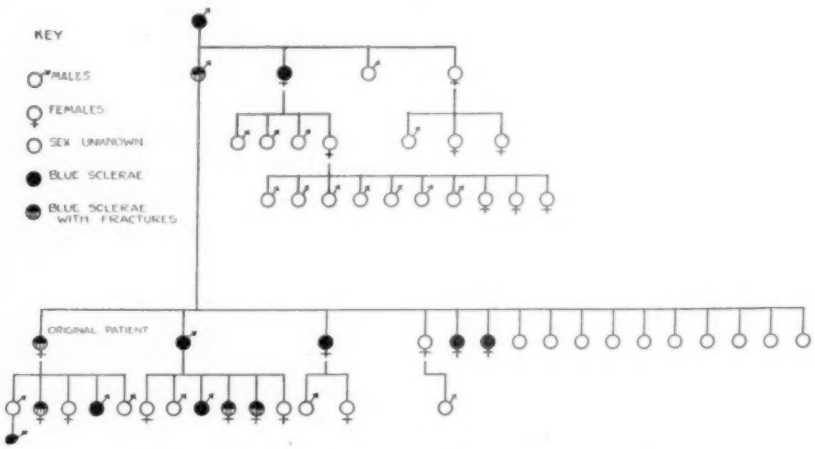

FIG. 7. Family tree of five generations of fragilitas ossium.

of the veins, indicating sclerosis, was noted in each of the three cases. The thinning of the shafts of the metatarsal and metacarpal bones producing relative enlargement of the head and the base, and giving the "dumb-bell" type bones, also warrants special mention. Studies of calcium in the blood were not conclusive. In one case oxaluria was reported.

Conclusions

Fragilitas ossium was traced through five generations of one family. It was associated with blue sclerae. Some of the cases sustained multiple fractures, others presented a distinct ligamentous relaxation, causing the condition known as double-jointedness. Phlebosclerosis was noted in all three of the cases studied.

We wish to thank Dr. Alfred F. Hess of New York for seeing these three cases with us, and Dr. M. Goldzieher and I. Sherman for the laboratory examinations.

A JUSTIFICATION OF THE DIAGNOSIS OF CHRONIC NERVOUS EXHAUSTION *

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"CHRONIC NERVOUS EXHAUSTION," a term synonymous in the minds of many persons with neurasthenia, psychasthenia, or neurocirculatory asthenia, lacks the clear-cut connotation of terms such as duodenal ulcer or pulmonary tuberculosis. The symptoms of chronic nervous exhaustion are protean; they may appear to originate in any or all bodily structures and systems. In its most common meaning, chronic nervous exhaustion indicates a long-present, subjective sensation of tiredness, disproportionately exceeding the effort which produces it and which cannot be accounted for by organic disease. Weakness, lack of energy and ambition, nervousness, unrestful sleep or insomnia, melancholia, tachycardia, and pains and aches in various parts of the body may be additional symptoms of the condition. The syndrome mentioned may be associated with organic disease, yet not occur as a direct result of it, but rather indirectly, as a result of the worries and uncertainties precipitated by organic disease that is recognized to be present by the patient.

The term chronic nervous exhaustion has little standing with many physicians; to them it is an admission of inability to indicate the situation or nature of an organic change that is responsible for the symptoms. To others the diagnosis is as significant and inclusive as that of chronic mitral endocarditis, for example. It is not germane to our subject to consider the respective viewpoints, although we accept chronic nervous exhaustion as an accurate diagnostic term. Nor is it the purpose of this presentation to consider the genesis of chronic nervous exhaustion, the means of distinguishing it from such conditions as psychoneurosis and constitutional biologic inferiority, or the manner of treatment of and the outlook for patients with this condition. It is well to keep in mind that the accuracy of the diagnosis of chronic nervous exhaustion should bear a direct relationship to the accuracy of the diagnosis of other conditions, depending on the caliber and equipment of the responsible physician or physicians.

For our investigation, we selected case records of patients examined at The Mayo Clinic; each acceptable case record was that of a patient who had been seen at the Clinic one or more times several years after the original diagnosis of chronic nervous exhaustion had been made. In these records we hoped to be able to trace the evidence of organic disease, if such were present, and to determine the tenability of the diagnosis of chronic nervous exhaustion. We assumed that if the clinical picture at the first examination

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was due to unrecognized organic disease, such organic disease should be detected by a subsequent examination or examinations over a period of years. The objections to such an assumption seem minor; the almost uniform tendency of organic disease is to progress, and to become, therefore, more obvious, or to improve and to lead to disappearance or diminution of the symptoms produced. Moreover, repeated examinations minimize the possibility of diagnostic error.

Material Studied

Our material consisted of the records of 235 patients who were reëxamined at The Mayo Clinic an average of six years after the original diagnosis of chronic nervous exhaustion had been made. In each case our study began with the original diagnosis of chronic nervous exhaustion. The entire number of patients may be divided into three groups, consisting of (1) patients who did not ever have organic disease, (2) patients who, at the time of the final examination, had organic disease that was not considered responsible for the original symptoms, and (3) patients who were found, subsequent to the time of the original diagnosis of chronic nervous exhaustion, to have organic disease which appeared to explain, at least in part, the symptoms originally complained of.

Group 1. (*Patients who did not have organic disease.*) This group is composed of 200 patients, 85 per cent of all patients studied. An average of about six and a half years elapsed between the original and the final examinations. The minimal period between the time of the first and last examinations was two years. No organic disease had developed subsequent to the original examination. It cannot be reliably concluded from this series of cases that patients with chronic nervous exhaustion fail to recover from the symptoms of which they complain. Doubtless many patients improve or are entirely relieved, and because of this do not return for reëxamination. Those who fail to receive the benefit mentioned are likely to return for further opinion regarding their condition.

Women comprised 78 per cent of the group, 25 per cent of whom were single and 6 per cent widowed or divorced. Forty per cent of the entire group of men and women were in the fourth decade of life, and about 20 per cent in each of the third, fifth and sixth decades of life. Thirty-four per cent of the women were housewives, and 15 per cent were teachers; a variety of occupations was recorded for the remainder of the women patients. Eight of the 44 men were farmers, five were physicians, four were lawyers and one was a dentist; the remainder of the group were of various occupations, such as merchants, bankers and laborers.

The duration of the symptoms in this group of cases ranged from three weeks to 46 years, and averaged about five years; in only 18 instances were the symptoms of shorter duration than one year.

It is difficult to give accurately the part of the body concerning which the patients complained when relating their symptoms; in most instances the

symptoms were multiple, and appeared to originate from more than one organ or system. The points of origin of the major symptoms were as follows: gastrointestinal tract, 69 cases; nervous system, 60 cases; muscular system, 37 cases; thyroid gland, 20 cases; and heart, 14 cases. The symptoms referable to the gastrointestinal tract were chiefly bloating and belching, soreness of the abdomen, constipation, eructation, and indefinite types of distress; those referred to the nervous system were nervousness, insomnia, headache, unrestful sleep, fatigue, emotional instability, and mental depression. The symptoms which apparently arose in the muscular system were weakness, fatigue, and indefinite aches and pains. Twenty patients complained of "goiter," and their chief symptoms were those listed as arising in the nervous system, or apparent enlargement of the thyroid gland. The chief symptoms referable to the heart were palpitation, tachycardia, and irregular rhythm. The following two cases are illustrative of the wide divergence in the type and duration of symptoms:

A woman, aged 50 years, had been in poor health most of her life; bloating, belching and abdominal discomfort had been present for 15 years. The same symptoms were present at the second examination 10 years after the original diagnosis of chronic nervous exhaustion had been made. Three major surgical procedures on the abdominal and pelvic organs had been carried out since the onset of the symptoms referred to the gastrointestinal tract; relief was not experienced.

A merchant, aged 56 years, had noticed dizziness and lack of energy following the death of his business partner three weeks previously; at that time his responsibilities and business cares had increased greatly. He was reëxamined 10 years later, at which time he stated that the symptoms noted at the time of his first examination had disappeared shortly after readjustment of his business.

This group of 200 patients had undergone a total of 289 separate operations; of these tonsillectomy accounted for 74. The remaining operations appeared to have been performed in most instances for relief of the same symptoms that were mentioned at the time the diagnosis of chronic nervous exhaustion was made at The Mayo Clinic. Eighty-one of the 156 women in this group of patients had undergone operations on the pelvic organs: uterine suspension, hysterectomy, plastic repair of cystocele and rectocele, or removal of oviducts, of ovaries or of ovarian cysts. Removal of the appendix, gall-bladder, thyroid gland, hemorrhoids, and surgical procedures on the accessory nasal sinuses accounted for the remainder of the operations. Failure to obtain relief in the majority of cases is indicated by the fact that the examinations at the Clinic were made on account of the symptoms for which the operations had been performed.

The points of significance in the records of the patients who comprise this group are the predominant proportion of women, the fact that a large proportion of the men were of "white collar" occupations, that the type and duration of symptoms were of wide range, and that numerous operations were performed for symptoms which were not relieved in the majority of instances.

Group 2. (*Patients who, at the time of the final examination, had organic disease that was not considered responsible for the original symptoms.*) There were 21 patients in this group which comprises 9 per cent of all cases studied. The patients were reëxamined at an average of seven years after the original examination. In all of the cases organic disease had developed (table 1). Patients were included in the group only if review of their

TABLE I

Summary of Final Diagnoses in Group 2

Diagnosis	Time, years*
Cholelithiasis (2 cases)	10 and 5
Duodenal ulcer (2 cases)	7 and 6
Carcinoma of breast (2 cases)	1 and 2
Carcinoma of uterine cervix (2 cases)	7 and 8
Menorrhagia	11
Gastric hemorrhage (?)	8
Leukemia†	7
Exophthalmic goiter (10 per cent possibility)	10
Cholecystitis (?)	9
Nontoxic adenomatous goiter (2 cases)	3 and 9
Pulmonary tuberculosis	10
Hypertension and arthritis	4
Bronchogenic carcinoma	10
Septicemia	6
Syphilis of central nervous system	5

* Between original and final diagnosis.

† Number of leukocytes in each cubic millimeter of blood was normal at the first examination.

records indicated that the eventual organic disease could not, in all probability, have been responsible in any degree for the symptoms originally noted. Although such a relationship cannot be proved or disproved with certainty, whenever doubt existed in our minds whether a certain patient should be considered in group 2 or group 3, the latter classification was used. In several instances in group 2, relationship of the organic disease to the original symptoms was disproved by the factor of time alone; carcinoma of the uterine cervix, leukemia, bronchogenic carcinoma, and septicemia could not have been present for seven, seven, ten, and six years, respectively, before their recognition. They could not, therefore, have been responsible for the original symptoms.

Group 3. (*Patients who were found, subsequent to the time of the original diagnosis of chronic nervous exhaustion, to have organic disease which appeared to explain, at least in part, the symptoms originally complained of.*) There were 14 patients in this group, comprising about 6 per cent of the entire number studied. The patients were reëxamined at an average of five years after the diagnosis of chronic nervous exhaustion had been made. All of them, except the patient with paroxysmal tachycardia, had organic disease at the time of the final examination. Although it is difficult to determine in any specific instance whether the organic disease

existed at the time of the diagnosis of chronic nervous exhaustion, consideration of the entire group leads us to believe that in some instances such was the case. It is impossible in most instances to determine whether the diagnosis of chronic nervous exhaustion was erroneous in the sense that closer examination would have revealed organic disease, or whether the organic disease was in such an early stage that it could not have been recognized clinically. We believe that in most instances in which the latter was true, the final diagnosis of organic disease accounted for the earlier symptoms.

Three conditions account for the organic diseases found in eight of the 14 cases in this group; namely, chronic encephalitis in three cases, hyperthyroidism in two cases and tuberculosis in three cases. The early symptoms of these three diseases tend to be vague or of little localizing value. Moreover, chronic fatigue is often one of the earlier symptoms of these conditions.

In two of the cases of chronic encephalitis the presence of organic disease was suspected at the time the diagnosis of chronic nervous exhaustion was made, but evidence of it was inconclusive. In the remaining case of this type, the disease was not suspected.

In both of the cases in which hyperthyroidism was finally recognized, it was suspected initially, because of the basal metabolic rate, but was not confirmed at that time.

One of the three patients who were finally found to have tuberculosis gave a history highly suggestive of tuberculous infection of the lungs, but the physical and roentgenographic examinations of the lungs did not confirm the suspicion. It is possible that pulmonary tuberculosis was present but unrecognized. In another of the cases of this type, either the diagnosis of healed tuberculosis at the primary examination was fallacious, or else reactivation of the infection had occurred. In the remaining case, pulmonary tuberculosis which apparently was suspected at the time of the original examination could not be demonstrated.

Six cases of the 14 remain to be accounted for. In the first of these, organic disease was suspected but not diagnosed at the time of the first examination; appendiceal abscess was finally discovered. Concerning the second case, it is impossible to state that syphilis of the central nervous system, which was recognized at the second examination, was responsible for the symptoms at the first examination. Careful survey of the records of the final examination of the third patient did not indicate sufficient reason to suspect the presence of typhoid fever, although seven weeks after the patient left the Clinic, we were informed, by letter, that she had died of this disease. The original diagnosis of chronic nervous exhaustion made 10 years previously appears to have been reliable. In the fourth instance, the diagnosis of chronic nervous exhaustion apparently was a frank error. Enlargement of the cervical lymph nodes was present, and two weeks later

this was found to be part of the syndrome of Hodgkin's disease. The original diagnosis in the fifth case may represent oversight, inasmuch as the urine contained leukocytes and erythrocytes; five years later, nephrolithiasis was discovered. The original diagnosis of chronic nervous exhaustion in the sixth case could well have been entirely reliable, but more than two years later definite paroxysmal tachycardia was recognized.

A summary of the 14 cases of group 3 shows that in only four instances did the original examiners fail to suspect the presence of an organic disease that almost certainly was responsible for the original symptoms. The paroxysmal tachycardia that appeared in another case was not caused by organic disease, but the original diagnosis of chronic nervous exhaustion may be considered in error inasmuch as it did not adequately explain the episodes of palpitation. These five cases represent errors in the diagnosis of chronic nervous exhaustion, if the term error be reserved for instances in which the examining physician was frankly at fault. They comprise only 2.1 per cent of the total number of 235 cases in which the original diagnosis of chronic nervous exhaustion was made.

Comment

If one considers that all cases of groups 2 and 3 represent errors in the diagnosis of chronic nervous exhaustion, the proportion of error is 15 per cent. If only the five cases of group 3 that have been mentioned are accepted as evidence of faulty diagnosis, and this seems more just, the proportion of error is 2.1 per cent. In other words, the greatest possible error in the diagnosis of chronic nervous exhaustion made under such circumstances as those presented is 15 per cent and the least error is 2.1 per cent. The percentage in our series of cases probably lies between the two mentioned; it appears to be about the 6 per cent represented by the 14 cases of group 3.

This impresses us as a gratifyingly small error when it is considered that the diagnosis of chronic nervous exhaustion cannot be made on the basis of objective findings but is, rather, based on the paucity of objective findings in addition to the results of careful study of the symptoms presented by the patient and the history elicited from him. It impresses us that a diagnostic accuracy of 94 per cent in chronic nervous exhaustion compares favorably with the accuracy of diagnosis in other conditions. However satisfying this percentage of accuracy may be, it is desirable to improve it. Our study shows that the conditions which need to be most carefully sought for, before the diagnosis of chronic nervous exhaustion is made, are hyperthyroidism, chronic encephalitis, and tuberculosis.

Errors in medical or surgical diagnosis are viewed differently. If a disease clinically diagnosed as acute appendicitis is shown at operation to be acute salpingitis, the diagnostician is considered much less in error than if a patient who appears to have chronic nervous exhaustion is eventually

shown to be affected with hyperthyroidism. Such discrimination is not only unfair, but it encourages the diagnosis of organic disease when none is present. Much is being said and written regarding nervous indigestion, constitutional biologic inferiority and the various types of psychoneurosis. A real service would be accomplished if the accuracy of such commonly made diagnoses can be determined.

Summary

Case records of 235 patients were studied in order to determine the accuracy of the diagnosis of chronic nervous exhaustion made on an average of six and a half years before final examination of the patient. In our cases, the accuracy of the diagnosis of chronic nervous exhaustion was found to be between 85 and 98 per cent. The actual figure seems to be about 94 per cent. The figure stated indicates that the diagnosis of chronic nervous exhaustion, when made under good circumstances, is reliable in high degree.

IRRADIATION TREATMENT OF HYPERTHYROIDISM *

By George E. Pfahler, M.D., Sc.D., F.A.C.P., *Philadelphia, Pennsylvania*

The internist is usually the first to come in contact with a case of hyperthyroidism. To him belongs the responsibility of making the diagnosis by its differentiation from other similar clinical syndromes; and he should remain in charge until the patient is restored to a normal condition and is able to carry on his usual occupation. The treatment of hyperthyroidism does not consist merely in treating the thyroid either by irradiation or operation; the predisposing and exciting causes of the disease should be removed, and after active local treatment the patient should be guided and guarded by competent medical advice. Crile,[1] says: "In 3.3 per cent of our total cases, there is a recurrence of the hyperthyroidism after partial thyroidectomy. In every case in which this occurs it will be found that there has been a persistence of the agents which were active in producing the primary hyperthyroidism—focal infection, social maladjustments, worry, overwork, or some other strain." In general, about 10 to 20 per cent of cases of hyperthyroidism fail to get permanently well, whether treated by surgery or irradiation. It is, therefore, quite proper that the subject of nonoperative treatment of hyperthyroidism should be presented before this body, for the general care of the patient is absolutely essential.

It is well known to you all that patients who are suffering from hyperthyroidism associated with a goiter postpone the consultation with their family physicians for fear that they will be sent to a surgeon for operation. To avoid such delays, and to conserve the patients' energy it is therefore well to remember that irradiation therapy is approximately of equal value to surgery in the end results, and if patients learn that not all cases must be operated upon, they will be less likely to delay consultation.

In dealing with hyperthyroidism, we must assume that there is an overgrowth or a new growth of the thyroid gland, or a hyperfunction of the normal amount of glandular tissue. The condition can be relieved by surgery, and satisfactory results can be obtained if the surgeon removes just the right amount of tissue; or the disease can be controlled by irradiation which gradually reduces the activity of the cells, even in cases with no tumor or hypertrophy, and causes an atrophy of the hypertrophic or hyperplastic tissue when present. With careful clinical observation checked by tests of basal metabolism, one need not exceed the necessary dosage.

Indications for Irradiation

Based upon our observations and those of other radiologists, we believe that irradiation is indicated in all cases of hyperthyroidism in which the

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patient is not in crisis, or is not suffering from definite pressure symptoms. We recommend operation in all simple or nontoxic goiters, unless there is some contraindication, in which case a moderate amount of irradiation may be used. Sometimes brilliant results are obtained even with large goiters of this type.

Advantages of Irradiation

1. The fear of operation is eliminated and therefore the patient is more likely to come under treatment early and before cardiac damage has taken place.
2. Due to elimination of this fear, and early treatment, the patients are not interrupted in their occupation. When the disease is well advanced, or serious symptoms are present, they must, however, be put at rest.
3. There is no pain or shock and no great inconvenience if the condition is treated reasonably early.
4. Patients with advanced disease or serious heart complications may be treated without shock, and if radium is used, need not even be removed from their room.
5. There is no risk of mortality from the treatment.
6. There is an absence of scars or keloid formation.
7. The end results are approximately equal to those obtained by surgery.

Objections

1. *Burns.* The danger of burns naturally comes into a patient's mind, because they have occurred occasionally in the early years of irradiation therapy when the method was being developed and, since such patients do not die, a few such accidents in the country become known far and wide, whereas if a patient dies during an operation, from any accident or any complication, burial takes place. It is accepted as one of the hazards of the procedure and may be ignored, or is soon forgotten.

2. *Telangiectasis and skin atrophy* are dangers which must be taken into consideration. In our cases the former has occurred in 3.0 per cent of 533 cases, but these have occurred in our earlier treated cases. Its occurrence depends upon the amount of the total irradiation, and while it may follow a single excessive dose of rays, it may occur without any erythema ever having been produced. We have not seen telangiectasis in any cases in which we had not given more than eight series such as are described in our technic. We aim, therefore, to obtain our results with from six to eight series. Even when telangiectasis has occurred, it has been, except in one case, only slight, and has not bothered the patients much.

3. *Exacerbation of symptoms* may occur after the first treatment. This has been especially emphasized by Borak,[2] Pordes [3] and Goette,[4] but in our experience such an increase has been insignificant and no more than may occur from time to time independent of treatment. If such an increase is feared, it may be well to decrease the dose in the first series, especially in the severe cases.

4. *Myxedema.* Myxedema has not occurred in any of our cases. Hypothyroidism has occurred only in four cases of our series, or less than 1 per cent, and in only one of these four cases was it sufficiently severe to require desiccated thyroid. Groover[5] reported subsequent hypothyroidism in only 1.3 per cent of his cases. Our low percentage of hypothyroidism after irradiation is in part due to our ability to reduce the excessive secretion gradually, and in part due to the fact that the normal thyroid cells are rather resistant to irradiation. Walters, Anson, and Ivy[6] state the experimental literature at hand indicates that the normal thyroid tissue is resistant to roentgen-rays and concluded from their own studies that the "normal thyroid of the dog is quite resistant to roentgen-rays and degenerative changes are not caused by the dosage used in the experiments, which is a dosage known to be of clinical value, and in the dosage used do not cause extensive proliferation of connective tissue." *We have also clinical proof that the normal thyroid is very resistant to irradiation, in the fact that we have obtained no hypothyroidism in any cases of carcinoma of the larynx, pharynx, or neck in which we have used many times the total dosage used in hyperthyroidism.* The diseased thyroid tissue on the other hand seems to be very sensitive as judged by clinical results.

5. *Damage to the Parathyroids.* Ivy and his associates state: "The experimental results indicate that the clinical dosage in the treatment of hyperthyroidism will not injure the parathyroids." Confirming the experimental work, we can state that none of our patients and none recorded in the literature have shown any tetany following irradiation. Neither has any tetany occurred following the enormous dosage used in the treatment of carcinoma of the larynx, in which the thyroids and the parathyroids are constantly exposed to irradiation of sufficient quantity to cause the surface skin and the surface of the mucous membrane to desquamate.

6. *Difficulty in Subsequent Surgical Removal if This Should Be Necessary.* This objection need only be considered in a small percentage of cases (only 6.8 per cent of our cases were operated upon), and in only one of these, an early case in our series, was any difficulty involved. It is now generally admitted that adhesions are found in as many cases which have had no previous irradiation as in cases which have been treated (G. Schwarz[7]). Eiselberg, who was first to consider adhesions as a postirradiation complication, now disregards them as irrelevant.

7. *Slow Response to Irradiation.* With irradiation we usually get some improvement at the end of a month and very definite improvement at the end of two months. If the surgeon uses two weeks preparatory to operation, two weeks to recover from the operation and a month for convalescence, the difference in time is not so great. At the end of these two months, however, we may generally expect a more complete relief of symptoms from surgery than from irradiation, because the surgeon removes the excess of glandular tissue at once, while with irradiation we reduce the hyperactivity and the hypertrophy or hyperplasia gradually. This is a slow and progressive effect.

This slow response is not without its advantages, however, since the activity of the gland can be checked by frequent metabolic determinations and the final result controlled with greater nicety than when the hyperfunctioning gland is removed at one sitting. During this period of early treatment the internist should use all his known methods of helping the patient.

8. *Permanent Cardiac Impairment Developing during the Prolonged Irradiation Treatment.* The improvement from irradiation comes more slowly than from surgery, but this difference is not as great as would at first appear. There is likely to be considerable delay and advancement of the disease and damage to the heart before the patient will consent to an operation, which will make up for any delay in the results from irradiation. Progress in cardiac impairment may also occur in cases treated surgically, as is indicated by the report made by Willius [8] from the Mayo Clinic, in which he states: "At the time of initial examination at the Mayo Clinic, auricular fibrillation was found in 7 per cent of patients with exophthalmic goiter, and in 9 per cent of patients with hyperfunctioning adenoma. These percentages are doubled while the patient is under observation, that is, during the preoperative, operative and postoperative periods. Auricular fibrillation may occur as a permanent, intermittent or paroxysmal disorder."

Holzknecht [9] states that not one case is found in the literature, proving that the prolonged period of irradiation resulted in unnecessary damage to the heart.

TABLE I

An Analysis of 698 Cases of Goiter *

Cases with hyperthyroidism treated with roentgen-rays	440
Cases with hyperthyroidism treated with radium	6
Nontoxic goiter cases treated	59
Malignancies of thyroid treated	28
Total cases treated	533
Simple or nontoxic goiters in which we advised against irradiation and which were not treated	165
Total	698

* These statistics have been collected from our records by my associate, Dr. Jacob H. Vastine.

Under hyperthyroidism or thyrotoxicosis we have classed all exophthalmic goiter cases, all toxic adenomas, and those which had a high basal metabolism associated with the characteristic nervous symptoms even when no goiter and no exophthalmos were present.

TABLE II

Results Obtained by Us in the Treatment of 440 Cases of Hyperthyroidism

	Cured	Markedly Improved	Not Improved
Percentage	57.3	30.6	12.1
Average number of treatments	6.1	5.7	3.7
Average time observed	6.5 yrs.	2.8 yrs.	
Total cured or markedly improved	87.9 per cent		

We have classed as cured those cases in which the basal metabolism is between plus 10 per cent and minus 10 per cent; the pulse has returned to normal; in which the weight has increased, approximately to what it was before the onset of toxic symptoms, and nervous and other clinical manifestations have subsided; and in which the goiter has either completely disappeared or is so involuted as to be entirely unobjectionable from a cosmetic standpoint.

We have classed as improved those cases in which the basal metabolism is within normal limits or markedly decreased; and in which all clinical signs of thyrotoxicosis have disappeared, except a residual myocardial deficiency, which was present before the beginning of irradiation. We have classed here also those inoperable cases given irradiation to reduce the toxicity and prepare them for operation. The nomenclature frequently used by the internists and surgeons is "economic restitution" or "rehabilitation." In our series it can be seen that in 87.9 per cent of the cases "economic restitution" or "rehabilitation" was obtained.

Recurrence

Among patients we regarded as cured there were only two post-irradiation recurrences, or less than 1 per cent. There were cases in which the metabolic rate was rapidly brought down to within normal limits and subsequent tests within several months showed it to have risen 5 or more per cent above normal. In such cases which had been insufficiently treated, one or two more series of treatments were necessary before stabilization of the metabolic rate permanently within normal limits was obtained. These were not regarded as recurrences, since the patients were really still under treatment.

Hyperthyroidism without Palpable Glandular Enlargement

This is a class of cases in which irradiation is particularly indicated. There were 37 of these cases in our series. There was economic restitution in 29. The treatment failed in five cases and three patients could not be traced.

In three cases which were not responding satisfactorily, we tried treatment over the cervical sympathetic ganglia and over the suprarenals but with no appreciable effects.

Hoarseness did not develop, because the larynx and arytenoids were protected. Four of our earlier cases developed a severe tracheitis, but these four had more than the eight series, and it was doubtful whether the effects were due to the irradiation.

No evidence of injury to the parathyroid glands was observed in any of our cases. It is likely that the normal parathyroids, like the normal thyroid, are not sensitive to irradiation. We believe, however, that a diseased parathyroid is radio-sensitive; a confirmatory clinical observation has been made recently upon this point by Merritt.[10]

Technic

We routinely employ roentgen-rays, using 130 kilovolts, 5 milliamperes, at 25 to 30 centimeters distance, with the equivalent of 6 millimeters of aluminum filtration. The cervical region is divided into four fields, approximately 5 by 15 centimeters in size. Two of these are anterior and two are posterolateral. The lower border extends down over the thymic region. The larynx is protected with lead. The rays are directed medially and downward, so that a cross-firing effect is obtained in the thyroid region. These four areas constitute one series and they are usually given in one day, repeating this series in three weeks, then in four weeks, increasing the interval according to the improvement obtained. In simple or colloid goiters, 30 or 40 per cent doses may be given through the four above fields without danger of producing hypothyroidism. These several small doses will often be sufficient to show a definite decrease in size. In adenomata, localized doses are usually employed, cross-firing the adenoma through two portals.

In the mildly toxic cases we give an initial 50 per cent skin erythema dose through each of four portals. This is repeated in three weeks and then the amount is decreased and the interval increased. In the severer cases it is better to begin with smaller doses, not exceeding 40 per cent at the first series. This may be increased at subsequent series. More than six series are rarely necessary, a good response sometimes being seen after four series. We are reluctant to give more than six or eight series. If a patient is not definitely improved after three to five series, and a lapse of two or three months from the beginning, other measures should be employed. A careful record of the patient's pulse, weight, and general health and the condition of the skin is made at each visit and frequent metabolic determinations are made. Foci of infection are removed. Chest examinations are made routinely on each new patient. We recommend administration of quinine hydrobromide in five to ten grain doses, three times a day, unless ringing of the ears occurs (Bram). Dodd's lotion is prescribed for application to the neck to avoid skin damage. The patient is cautioned against sunburning the neck or applying irritating salves or lotions. Rest, so far as practicable, is advised, the patient being told never to stand when she can sit and never to sit when she can lie down. A high caloric diet of easily digested food is recommended. The patient is advised against the use of stimulants. We have not found the administration of iodine helpful.

We employ roentgen irradiation routinely, because it has produced such uniformly good results. We have treated only a few cases with radium which were unsuitable for roentgen-ray treatment. Loucks [11] and Ginsburg [12] prefer radium and have obtained excellent results. Surely as good results can be obtained with radium as with roentgen-rays. It would seem that radium is preferable for patients who cannot come for treatment, or for whom the excitement caused by the machinery would be harmful. We have successfully used radium needles, interstitially, in a localized adenoma

which did not respond to roentgen therapy as fast as we felt it should. This can be done under mild narcosis without shock to the patient and has proved to be a valuable procedure in selected cases.

Results Obtained by Other Radiologists

Our results closely parallel those of other radiologists. In a previous presentation we reviewed the reports of 20 other radiologists, covering over 3300 cases, of which an average of 85 to 90 per cent have been cured or markedly improved. There have been several excellent reports upon this subject since that time. That of Menville [13] is probably the most comprehensive and the most representative, since he reviewed the work of 75 radiologists, both in Canada and the United States, by sending questionnaires. This report covered 10,541 cases treated by irradiation, of which 66.2 per cent were cured, 21 per cent were markedly improved and 12.4 per cent were not improved. It is interesting to note that 10 per cent of these 10,541 were cases in which surgery had previously been tried without success. There were 8.45 per cent of recurrences following irradiation. These results, which are a fairly accurate cross-section of the work done by radiologists generally, compare favorably with the cross-section of the work done by the average surgeon as reported by MacLean [14] in which he found the operative mortality rate alone to be 7 per cent in cases of exophthalmic goiter treated surgically, although the best clinics report less than 1 per cent.

Brief Reports of Cases Having No Definite Thyroid Enlargement

It is impractical in a paper of this kind to make a detailed record of all cases. Probably the most interesting and most difficult cases for diagnosis are those in which there is no definite palpable thyroid enlargement, or in other words, no goiter. Hamburger and Lev,[15] in 1930, have reported a series of such cases and again called attention to the difficulty of making a diagnosis of hyperthyroidism in the absence of goiter and in the absence of other typical symptoms. They referred to the fact that Charcot [16] recognized this difficulty in 1885, and Chvostek [17] also in 1887. Further studies were also made on this group of cases by Levine and Sturgis,[18] and Priest [19] and Tucker.[20] It is this group of cases that are particularly liable to be overlooked and mistreated for a long time. It is also this group of cases which are probably less favorable for operation. We are likely to see more of this class of patients, because of the fact that no goiter can be felt, and they are therefore referred to radiologists for treatment. I am, therefore, recording very briefly in chronological order, our cases which had no definite thyroid enlargement, as they have been collected from my office records for me by my associate, Dr. Jacob H. Vastine. Those patients, referred for treatment, had been studied and diagnosed by eminent clinicians whose diagnosis I accepted as the best that could be made.

Case I

Miss M. K., age 19, was referred for treatment of exophthalmic goiter on April 5, 1913. The patient developed this condition after an attack of influenza in December 1912, after which she became extremely nervous, and had a rapid pulse. She was weak. Her eyes were strikingly prominent, and while she had some general fullness of the neck, there was no palpable goiter, or definite localized enlargement. Her general appearance, however, was that of a typical exophthalmic goiter. She was given fractional doses of roentgen-ray treatment, which was the custom at that time, between May 5, 1913 and June 26, 1913. On October 7, 1914, she was free from all symptoms, except the prominence of her eyes. She has remained free from symptoms, except for the eyes, which have not yet returned to normal, though they have shown marked improvement. Since then, she has been married. She was in an airplane accident in 1928, after which her nervousness increased somewhat, but an examination on October 5, 1929 showed no evidence of goiter, and only slight exophthalmos. Her skin was normal; her weight 147 lbs.; her pulse at rest 78, and after exercise 84. She was reported well on January 13, 1933.

Case II

Miss A. R., age 28, referred on May 28, 1918, by Dr. L. N. Boston, of Philadelphia, for treatment of hyperthyroidism. Exophthalmos had been present about six years, and one and a half years previously both thyroid arteries had been ligated by Dr. A. C. Wood. There was no enlargement of the thyroid. She improved temporarily after the ligation. She was brought for treatment on a stretcher. Her pulse was 136 while lying down. She was extremely nervous. She showed definite improvement after the first treatment, and after four treatments, she was able to sit up for three hours at a time, and could walk a distance of a city block without resting. In April 1919, she returned to work in a knitting mill. She was given eleven treatments in all, during a period of eleven months. On January 28, 1929, her pulse was 68 at rest, and 78 after exercise.

Case III

Mrs. E. S., age 25, was referred on April 30, 1919, by Dr. Wm. H. Good, of Philadelphia, for treatment of hyperthyroidism associated with exophthalmos, but without any enlargement of the thyroid gland. At rest, her pulse was 80, but after slight exertion, was 120. We gave four series of treatments with the roentgen-ray during a period of three months, after which her nervous symptoms had definitely improved. She was reported entirely well by Dr. Good on July 14, 1929, or approximately ten years after beginning treatment.

Case IV

Mrs. F. E., age 32, was referred for treatment of hyperthyroidism by Dr. C. M. Fish, of Pleasantville, N. J., on May 25, 1920. Four years previously she developed a profound and unexplained asthenia, and she began to feel tired without doing anything. It even made her tired to go downhill. During the year prior to treatment, she had had a persistent tremor. She had been treated during a year and a half for rapid heart by Dr. Carrington of Atlantic City. She then entered the University of Pennsylvania Hospital, March 1, 1920, under the care of Dr. H. M. Fussel who made a diagnosis of hyperthyroidism. She rested in bed for several weeks. The basal metabolic rate was plus 15. There was no palpable goiter, and no exophthalmos. She suffered from attacks of diarrhea. Dr. Fussel advised ligation of the thyroid artery. An abnormal shadow was shown by the roentgen-rays in the upper mediastinum which probably was an enlarged aberrant thyroid. She showed some improvement from rest in bed before coming to me. At the beginning of roentgen-ray treatment, the

pulse was 120. On January 7, 1921, all her symptoms had disappeared, and she was apparently well. She was given six series of roentgen-ray treatments between March 25, 1920 and October 29, 1920. The basal metabolic rate in July 1931 was plus 6. On March 20, 1933, her pulse on arrival was 80, after exercise it arose to 90. At this time, her hyperthyroid symptoms seemed to have entirely disappeared.

Case V

Mr. A. T. B., age 39, referred by Dr. T. E. Wills of Pottstown, Pa. on June 30, 1920, for roentgentherapy for hyperthyroidism. During six months, he had little or no enlargement of the thyroid, but he had tremors, nervousness, palpitation, loss of weight, and tachycardia. Roentgen-ray examination showed a substernal thyroid enlargement. Under roentgen-ray treatment, all of his symptoms disappeared, and on October 6, his pulse was 78 after exertion, and he was practically free from symptoms. On December 17, 1920, he was completely free from symptoms. Dr. Wills reported him well on January 26, 1933.

Case VI

Mrs. M. W. was referred by Dr. A. F. Collier, of Waterbury, Conn., for treatment of hyperthyroidism on January 31, 1921. Her previous treatment had consisted of rest and iodine treatment which gave some temporary improvement, but the symptoms recurred, and there was no improvement during a period of six months. The symptoms consisted of nervousness, excitability, palpitation, exhaustion, loss of weight, and some exophthalmos. The thyroid was not enlarged. The patient was given nine series of roentgen-ray treatments between January 31, 1921 and May 17, 1922. There was improvement after the first treatment, and at the time of the last treatment, her pulse was 90 as compared with 140; her weight was 153 as compared with 131; her basal metabolic rate was plus 3.

Case VII

Miss E. P., age 28, was referred on account of hyperthyroidism, with no palpable goiter, on March 30, 1921, by Dr. E. H. Goodman, and Dr. John H. Musser of Philadelphia. The basal metabolic test showed plus 24. Her pulse rate was 136. Roentgen-ray examination showed no enlargement of the thyroid. The heart action fluoroscopically was very excitable. An examination of the eyes showed some congestion in the left eye, but no exophthalmos, and nothing else abnormal. Roentgen-ray examination of the pituitary showed some calcareous deposit in the region of the sella. After her second series of treatments, she felt very much better. She was less nervous, but still had some of the former vague fullness and distress at the back of the head which, however, was less pronounced. Her pulse had dropped to 100. The last treatment was given October 20, 1921, at which time she seemed to be very much better in all her symptoms. However, she developed some new symptoms in December and was referred to Dr. Ernest LaPlace by Dr. Goodman for operation upon the thyroid. She was finally sent back October 12, 1922 for an examination of her spine on account of severe pains. The roentgen-ray examination showed hypertrophic osteoarthritis, and at this time her hyperthyroid symptoms had returned even after the operation, so that she was too nervous to permit a roentgen-ray examination of her teeth. This case is marked as a failure, but it was also a surgical failure.

Case VIII

Dr. R. H. B., age 38, came of his own accord on May 4, 1921. He had been discharged from the army on December 19, 1919, with a diagnosis of toxic goiter, and valvular heart disease. He knew of no symptoms of heart disease except that he got very tired, was extremely nervous, and had a rapid heart. Dr. Crotti of Columbus,

Ohio, had seen the patient and had made a diagnosis of hyperthyroidism. He found no enlargement of the thyroid. The duration of his symptoms was 18 months. He suffered from nervousness, sweating, loss of weight, tremor, and exophthalmos. He received four series of roentgen-ray treatments from May 4, 1921 to August 11, 1921, at which time his symptoms had disappeared. On January 9, 1933, the patient reported: "General health good. No other treatment. Pulse after rest 70; pulse after exercise 86. Weight 137 in the nude. Neck normal. Eyes normal."

Case IX

Mrs. H. M., age 24, was referred on May 7, 1921 by Dr. Andrew Jackson, of Waterbury, Conn., for roentgentherapy of hyperthyroidism which had followed pregnancy two years previously. She had developed a rapid heart, a general nervous condition, and loss of weight, but she had no exophthalmos, and no enlarged thyroid. The cardiac action had the excitability as seen fluoroscopically which is characteristic of what one finds usually in hyperthyroid cases, and which, while probably not pathognomonic, at least always makes me think of hyperthyroidism. Her pulse at rest was 104, and after exercise 130. This abnormal increase in pulse rate with slight exercise seems to run parallel with the basal metabolism as determined in more recent years. She was given nine series of roentgen-ray treatments between May 7, 1921 and February 18, 1922, and on this date, a basal metabolism test was made which was minus one. She had increased in weight from 113.5 to 135 lbs. All of her other symptoms had disappeared. She was reported well January 31, 1933.

Case X

Mrs. J. S., age 33, was referred by Dr. Andrew Jackson, of Waterbury, Conn. on May 21, 1921, with the diagnosis of hyperthyroidism. She had been nervous for many months, but one month before coming to me, she had difficulty in swallowing, a feeling of suffocation, and a constant oppression in the throat. This led to a suspicion of a goiter, but Dr. Jackson and I were unable to palpate a goiter. On the basis of loss of weight, tachycardia, and her general nervous condition, he made a diagnosis of hyperthyroidism. I was unable to palpate any enlarged thyroid, but by roentgen-ray examination, I found a compression of the trachea, and a diagnosis of substernal thyroid was made. Five series of roentgen-ray treatments were given between May 21, 1921, and November 25, 1921, at which time her symptoms were relieved; she looked well, felt fine, and her pulse had returned to normal.

Case XI

Miss V. E., age 29, was referred by Dr. T. H. Weisenburg, of Philadelphia, for treatment of hyperthyroidism, on June 29, 1921. Previously she had had a double ligation in June 1916 and thyroidectomy in 1917. Before coming to us she had been suffering from toxic symptoms and had been bedfast for six months. She had definite exophthalmos but no palpable thyroid. She was given four series of roentgen-ray treatments between June 29, 1921 and October 12, 1921, at which time she was free from symptoms and was delighted. She immediately took up training as a technician under me. She completed this course as roentgen-ray technician, and then served as an assistant technician for an additional year in our hospital when she left to accept a position in another hospital. Her present address has been lost.

Case XII

Miss M. L. O., age 49, was referred by Dr. Judson Daland of Philadelphia, on April 29, 1922, for treatment of exophthalmic goiter, of about one year's duration. The thyroid, however, was not enlarged. She had nervousness, irritability, sweating, dyspnea, palpitation, and loss of weight, associated with tremor and exophthalmos.

Her basal metabolic rate was plus 40. She was given nine series of treatments and on April 16, 1923, Dr. Alex. Klein wrote as follows: "This patient was referred to me by Dr. Daland in May of last year and has been under my care since. Her basal metabolism at that time was plus 40, pulse 90, respiration 24, systolic blood pressure 155, diastolic 100. A second test on November 27, 1922, shows a basal metabolism of plus 30, pulse 92, respiration 28, systolic pressure 160, diastolic 100. A test made two days ago showed a basal metabolism of plus 7, pulse 62, respiration 19, systolic pressure 170; diastolic 110. The basal rate is normal. This very marked improvement is due entirely to roentgen-ray treatment. The increasing hypertension is most probably a result of her menopause." On May 22, 1933, this patient called for inspection and was entirely well. Her pulse was 68, and her weight 153.

Case XIII

Miss E. J. H., age 39, was referred for roentgen-ray treatment on account of hyperthyroidism on November 2, 1922 by Dr. Julian Adair, of Wilmington, Del. She was highly nervous and would break down when she attempted to do a full day's work of eight hours. She had not been able to work for five months. She had sweats, diarrhea, irritability, palpitation, tremor, but no goiter and no exophthalmos. She had a wild excited appearance. Her basal metabolic rate was plus 45. The pulse rate during rest was 130; after exercise 150. She was given the first series of roentgen-ray treatments on November 3, 1922. The second series was interfered with by an automobile accident and therefore could not be given until December 8, 1922. On August 5, 1929, the patient was symptomatically well. Her pulse was normal.

Case XIV

Mrs. W. H., age 47, was referred by Dr. Edwin H. Johnson, of Naugatuck, Conn., on May 9, 1923 for treatment of exophthalmic goiter. Her eyes had always been prominent, but during the previous 18 months this prominence had definitely increased. She had the appearance of being extremely nervous; she was irritable and had sweating of the skin, asthenia, and tachycardia. Her pulse was 140, her basal metabolic rate was plus 24. We could find no enlargement of the thyroid. She had eight series of roentgen-ray treatments between May 9, 1923 and April 6, 1924. Her basal metabolic rate on December 18, 1924 was plus two. The exophthalmos persisted at least until July 1, 1929, at which time she was in other respects well.

Case XV

Mr. H. A. D., age 54, was referred for treatment of hyperthyroidism by Dr. M. E. Alexander, of Waterbury, Conn., on August 15, 1923. The duration of his symptoms was three years, but they had been worse during the previous year. They consisted of marked nervousness, definite exophthalmos, loss of weight, without enlargement of the thyroid, but with asthenia, marked tremor and a pulse varying between 130 and 140. The following note was made by Dr. Alexander on July 31, 1923: "I looked over Mr. D. very carefully and came to some very definite conclusions. The patient is suffering from Graves' disease, but his case presents features which make me firmly believe that operation is contraindicated. First, his thyroid gland is not enlarged, barely palpable. His case is not due to poisons in the system, elaborated by an enlarged thyroid, but is part and parcel of a disturbance of internal secretion. Removal of the thyroid would only combat part of the trouble. Since the thyroid is not enlarged, the operation is often more difficult, and the results less certain. We must also take into consideration the condition of his heart, which is really far from normal. Certainly one cannot say that he can stand an operation with impunity." The basal metabolic rate on July 31, 1923 was plus 22. Fluoroscopic examination showed no goiter, or intrathoracic enlargement, but the heart showed enlargement of

both the right and left chambers. An electrocardiogram showed evidence of left preponderance and a heart rate of 130. He was given two series of roentgen-ray treatments, the second one September 13, 1923. The trip, however, was so exhausting and he was in such serious condition that we hesitated to give him further treatment. We advised treatment nearer home. On December 8, 1923, we received the following letter from Dr. Alexander: "Sorry to report that the patient died two weeks ago. The terminal illness was cardiac decompensation and hypostatic congestion of the kidneys. It is really no wonder that he died since he was in such an extremely poor condition when I sent him to you. You will please recall that I saw him only a few days before sending him to you." (Marked as a failure.)

Case XVI

Mrs. W. H., age 64, referred by Dr. G. M. Piersol, of Philadelphia, on March 28, 1924, for treatment of hyperthyroidism. The basal metabolic rate was plus 60. She was extremely nervous and at times had diarrhea. She had lost about 40 pounds of weight and suffered from asthenia and tachycardia. Her pulse varied between 110 and 140. There was no palpable goiter, and no exophthalmos. She received eight roentgen-ray treatments between March 28, 1924, and December 16, 1924. Her basal metabolic rate on February 17, 1925, showed plus 5, and she was free from symptoms. She was still well on June 25, 1929.

Case XVII

Mr. C. P., age 35, was referred for treatment of hyperthyroidism on November 20, 1924, by Dr. T. H. Weisenburg, of Philadelphia, at which time the basal metabolic rate was plus 48. There was a history of toxic symptoms during two to three years, consisting of nervousness, irritability, sweating, tachycardia, asthenia, and associated with hallucinations. There was no enlargement of the thyroid. The patient received only one series of roentgen-ray treatments. Then he moved to New York and later to Europe. According to the last report on January 19, 1933 his general health was fair. He had no other form of treatment. (Marked as a failure.)

Case XVIII

Miss B. C., age 22, was referred for treatment of hyperthyroidism by Dr. Marika Lambichi, of Chester, Pennsylvania on December 3, 1924, at which time her basal metabolic rate was plus 18, and her pulse 110. Her symptoms had existed since July 1924. She was markedly nervous, irritable, had abnormal sweating, and palpitation of the heart. There was no palpable thyroid, but there seemed to be an enlargement of the upper mediastinal shadow, suggesting a substernal thyroid. The cardiac action was of the type that one sees in hyperthyroidism as shown fluoroscopically. She was given six series of roentgen-ray treatments, and on June 22, 1924 she was free from symptoms and her basal metabolic rate was zero. Her basal metabolic rate on July 9, 1931 was plus 6. She was well on February 17, 1932.

Case XIX

Mrs. H. W., age 68, was referred on April 25, 1925 by Dr. Gordon Saxon and Dr. Earl French, of Philadelphia, for treatment of hyperthyroidism associated with cardiac dilatation. The patient had a rapid pulse, weakness, and nervousness, but no enlargement of the thyroid, and no prominence of the eyes. She had marked tachycardia, with a pulse of 134 when at rest, and 160 after exercise, associated with the peculiar cardiac action as seen fluoroscopically which one finds in hyperthyroidism, and which often leads us to suspect hyperthyroidism. The roentgen examination of the chest showed a strong suggestion of enlargement of the middle lobe of the thyroid downward, but there was no palpable enlargement. The cardiac shadow was in-

creased. We gave her four series of roentgen-ray treatments between April 25, 1925 and September 12, 1925, at which time she was markedly improved, and Dr. French reported on July 22, 1929 that the patient was well. He regarded this as a remarkable cure.

Case XX

Mrs. M. H. H., age 62, was referred by Dr. Wm. S. Bertolet of Reading, Pennsylvania on September 19, 1925. She had, however, been treated about 10 years previously at the Medico-Chirurgical Hospital for exophthalmic goiter. Her chief complaints were tachycardia, slight exophthalmos, and extreme nervousness, loss of weight, palpitation of the heart. Her pulse was 140, and her basal metabolism was plus 47. We gave her nine series of roentgen-ray treatments between September 19, 1925 and June 8, 1926, at which time her pulse had returned to normal. Her nervousness had disappeared. Her weight had returned to normal. She was seen on May 16, 1927, when she appeared well. When seen on February 26, 1932, her pulse was normal, 76 at rest, and 84 after exercise. On February 26, 1932, she was well, working regularly as a teacher.

Case XXI

Miss F. M. B., age 44, was referred for treatment of hyperthyroidism on October 29, 1925, by Dr. H. M. Eberhard, of Philadelphia. The basal metabolic rate determined on October 27, 1925, was plus 36. She had been treated in the Woman's Homeopathic Hospital for a nervous breakdown two years previously. She was kept at rest in bed for one month and this was followed by improvement. One of her chief complaints was a "dizziness confined to the left side of the head and left side of the body." In addition to this, she complained of nervousness, irritability, sweating, palpitation and a rapid pulse. She had no thyroid enlargement. We gave her three series of roentgen-ray treatments between October 29, 1925, and December 31, 1925, at which time she seemed to be well. The basal metabolic rate on December 27, 1925 was minus 3. She was reported well by Dr. Eberhard on July 16, 1929.

Case XXII

Mrs. W. P. N., age 39, was referred by Dr. Gordon Saxon, of Philadelphia, April 20, 1926. Her basal metabolic rate was plus 68. The duration of symptoms was six months. Her chief symptoms were nervousness, dyspnea, palpitation, sweating, loss of weight, and marked tremor. There was no thyroid enlargement. The patient was emaciated in appearance. Her pulse varied between 128 and 150. She was given seven series of roentgen-ray treatments between April 20, 1926, and November 15, 1926, at which time, she seemed to be well. On January 27, 1927, Dr. Saxon did a test of basal metabolism which showed plus 10. Her weight had increased from 104 to 124; her pulse had dropped from 150 to 92. The basal metabolism record in her case was as follows: April 20, 1926, plus 68; June 5, 1926, plus 25; September 7, 1926, plus 19; January 24, 1927, plus 10; February 12, 1932, plus 1. She was reported well on January 20, 1933.

Case XXIII

Miss M. F., age 47, was referred by Dr. Carl F. Welden, of Bethlehem, Pennsylvania, on November 7, 1927, for roentgen-ray treatment of hyperthyroidism. Her chief complaints were nervousness, weakness, and loss of weight. Her basal metabolic rate was plus 36. The pulse at rest was 126, after exercise 143. According to Dr. Welden, at times the pulse went as high as 200. She had no appreciable enlargement of the thyroid, and no exophthalmos, but she had tachycardia, asthenia, loss of 19 pounds in weight. We gave her seven series of treatments between November 7, 1927 and April 21, 1928 at which time her pulse at rest was 80, and after exercise 106.

On February 29, 1932, her pulse was 76 at rest, and 82 after exercise. She had gained 27 pounds.

Case XXIV

Mrs. R. C., age 31, was referred for treatment of hyperthyroidism by Dr. Clifford Waas, of Atlantic City, N. J., on April 25, 1928. Her symptoms had extended over a period of a year and a half, consisting of nervousness, irritability, palpitation, loss of weight, dyspnea, and tremor. Her basal metabolic rate was plus 16, on April 25, 1928. I was unable to find any enlargement of the thyroid by roentgen-ray examination or palpation, and no exophthalmos. She had two series of roentgen-ray treatments April 25, and May 16. When examined on July 20, 1929, she was free from any symptoms, had gained 15 pounds in weight and seemed to be in perfect health. She was reported well on January 5, 1933.

Case XXV

Miss T. B., age 48, was referred by Dr. Francis Dever, of Bethlehem, Pennsylvania on May 14, 1928, for treatment of hyperthyroidism. She had extreme nervousness during three months with loss of 40 pounds in weight. She suffered with marked tremor, excitability, and had a pulse rate of 145. She had no goiter, and no exophthalmos. Her basal metabolic rate was plus 40. She was given six series of roentgen-ray treatments between May 14, 1928, and October 2, 1928, at which time she was free from symptoms, and had gained 15 pounds in weight. Her pulse at rest was 88, and after exercise 98. Her basal metabolic rate was minus 7. She was well July 10, 1929.

Case XXVI

Mr. C. S. H., age 45, was referred by Dr. J. M. Anders and Dr. John H. Dripps, of Philadelphia, on June 2, 1928. There was a history of thyroid enlargement since February 19, 1928, but at the time the patient came to us there was no palpable enlargement. There was no exophthalmos. He had lost 27 pounds in six weeks, had marked tremor, was very irritable, suffered from dyspnea, palpitation, diarrhea, and the basal metabolic rate was plus 97. This patient came for only one treatment and was then operated upon by Dr. Charles Frazier in July and in October 1928. (Marked as a failure.)

Case XXVII

Miss M. P., age 19, was referred for treatment of hyperthyroidism by Dr. Horace Ewing, of Philadelphia, on July 12, 1928. Her chief symptoms were loss of weight, nervousness, asthenia, and a basal metabolic rate of plus 58. Her pulse rate at rest was 104, and after exercise 120. The patient improved after the first treatment but came only for one more. The final result is unknown. She cannot be traced.

Case XXVIII

Mrs. W. B. F., age 46, was referred for treatment for exophthalmic goiter, on March 18, 1929, by Dr. Thomas Klein, of Philadelphia. The patient had a thyroidectomy done four and three-fourths years previously, and also had had a few roentgen-ray treatments, following which she felt fairly well for one year, and then became nervous again, lost weight, and developed an exophthalmos of the left eye. She had the appearance of being poorly nourished, and had a noticeable exophthalmos of the left eye. Her basal metabolic rate was plus 35. The patient had two series of roentgen-rays (added to that already received before coming to us) on March 18, 1929, and April 10, 1929. She has had no treatment since, because the last treatment given to her gave some symptoms of indigestion which she blamed on the treatment, and therefore, she was unwilling to have more. On January 16, 1933, her weight had increased two pounds, her neck was normal, and her eyes were normal, but her pulse was still

120. She had insufficient treatment, but there seemed to be some improvement as a result of the treatment she had received. (Marked as a failure.)

Case XXIX

Mrs. H. S., age 43, was referred for treatment of hyperthyroidism by Dr. Myrtle Frank, of Egg Harbor, N. J., on March 27, 1929. The patient had only one treatment. She had symptoms of nervousness, but no palpable thyroid enlargement. She had irritability, palpitation, dyspnea, and no exophthalmos. She complained of tremor and sweating. Her basal metabolic rate was plus 20. She could not be traced.

Case XXX

Mrs. A. J. S., age 32, was referred for treatment of hyperthyroidism on December 2, 1929, by Dr. F. A. Faught, of Philadelphia. Her chief complaint was nervousness, loss of 38 pounds in weight, asthenia, headaches, pain in the back of the head and neck intermittently. There was doubtful bilateral enlargement of the thyroid. She complained of some palpitation, but no exophthalmos. She had slight tremor. Her basal metabolic rate was plus 20. The patient received five series of roentgen-ray treatments between December 2, 1929 and May 19, 1930, when the basal metabolic rate was plus 7, the pulse 82, and her general health improved. Her basal metabolic rate in March 1931 was minus 4. She had no fullness in the neck, and her eyes were normal.

Case XXXI

Miss E. B. B., age 23, a stenographer, was referred on February 19, 1930, by Dr. E. W. Pangburn, of Philadelphia, for treatment of hyperthyroidism. During six months, she had complained of extreme nervousness, irritability, sweating, asthenia, loss of weight, and palpitation. Her eyes were very prominent; her pulse was 120. Fluoroscopically, the heart action showed the excited action which always suggests hyperthyroidism. No goiter was palpable, and no goiter was demonstrated by roentgen examination. Her basal metabolic rate was plus 15. She was given three series of roentgen-ray treatments between February 19, 1930, and April 10, 1930. The basal metabolic rate on June 5, 1930, was plus 3. Her pulse was normal, but her eyes were still slightly prominent. On January 18, 1933, she was well.

Case XXXII

Miss F. G., age 21, was referred for treatment of hyperthyroidism by Dr. L. F. Luburg, of Philadelphia, on March 28, 1930. In December 1927, she had a basal metabolic rate of plus 4. In December 1929, she developed definite symptoms of hyperthyroidism and her basal metabolic rate was plus 41. Under iodine treatment, this dropped on February 6, 1931 to plus 13. On March 3, 1930, her basal metabolic rate was again plus 21. Her chief complaints were "peculiar waves" passing over her entire body, at times in her face, and other times in localized areas through her body. In addition to these nervous symptoms, she had tachycardia and asthenia. There was no prominence of the eyes, no enlargement of the thyroid. We made a roentgen-ray examination but could recognize no substernal enlargement, and no shadow that could be interpreted as an enlarged thyroid. Her basal metabolic rate on April 3, 1930, was plus 63. She received seven series of roentgen-ray treatments between March 28, 1930 and June 12, 1930, when she seemed to be symptomatically well. Her basal metabolic rate on July 23, 1930, was plus 2. She was reported well on January 18, 1933.

Case XXXIII

Dr. M. L., age 25, a dentist, was referred on April 15, 1930, by Dr. Francis J. Dever, of Bethlehem, for treatment of marked hyperthyroidism. His basal metabolic

rate was plus 42. The patient had exophthalmos and some general fullness of the neck, but no goiter. He had seven series of roentgen-ray treatments between April 15, 1930 and October 27, 1930. He was not responding well. Therefore, treatment was given on December 8, 16, 27, and 30, 1930, over the suprarenals. The basal metabolic rate dropped to plus 26, but he was not well. The patient was operated upon on April 30, 1931. There has remained some prominence of the eyes, but otherwise the patient has recovered. (Marked as a failure.)

Case XXXIV

Miss A. S. K., age 38, librarian, was referred for treatment of hyperthyroidism by Dr. Gordon Saxon of Philadelphia, on May 16, 1930. At that time her basal metabolic rate was plus 74. This condition developed after an attack of influenza in January 1930. Her chief symptoms were nervousness, irritability, palpitation, asthenia, dyspnea, and loss of weight. There was no enlarged thyroid, and no prominence of the eyeballs. Her pulse rate was 126. She was given eight series of roentgen-ray treatments between May 16, 1930 and January 30, 1931. Her basal metabolic rate on January 30, 1931, was plus 33. On September 19, 1931, it was plus 28 and on March 21, 1932, it was plus 10.

Case XXXV

Mr. C. E. W., age 57, was referred for treatment of hyperthyroidism by Dr. Francis J. Dever, of Bethlehem, on November 29, 1930. He was suffering from exophthalmos, tickling sensation in the throat, marked nervousness, irritability, palpitation, sweating, loss of weight, and tremor. He had a basal metabolic rate of plus 70, with no enlargement of the thyroid. He improved clinically, but not sufficiently. His basal metabolism did not improve. It was still plus 71 on May 13, 1931. He was, therefore, operated upon by Dr. Wm. L. Estes, Jr., who did a subtotal thyroidectomy, after which he recovered except for the prominence of his eyes. (Marked as a failure.)

Case XXXVI

Mrs. J. B., age 61, was referred for treatment of hyperthyroidism by Dr. Gordon Saxon, of Philadelphia on November 2, 1931. The duration of her symptoms was seven months. They consisted of nervousness, asthenia, palpitation, and loss of about 32 pounds of weight. She had tachycardia with a pulse rate at rest of 140 and after exertion of 156. Her basal metabolic rate was plus 33. She also had a marked tremor. Roentgen-ray examination of the chest seemed to show some increased substernal shadow above the arch of the aorta, which might be due to enlarged thyroid, but there was no palpable thyroid enlargement and no exophthalmos. We gave her three series of roentgen-ray treatments between November 2, 1931 and January 4, 1932. On June 13, 1933, she was reported as being entirely well by Dr. Saxon.

Case XXXVII

Mrs. T. M., age 35, was referred by Dr. Gordon Saxon, and Dr. William Higbee, of Philadelphia, on May 27, 1932, for treatment of hyperthyroidism. The patient had had a thyroidectomy on March 19, 1927. During the year before coming to us, she was extremely nervous, irritable, had palpitation, asthenia, sweating, loss of weight, occasional diarrhea, and tachycardia. Her basal metabolic rate was plus 13. The patient received four series of roentgen-ray treatments between May 27, 1932 and July 17, 1932, at which time she had shown marked improvement, the pulse rate being approximately normal and her weight having increased. The basal metabolic rate on October 8, 1932, showed minus 20. At that time her pulse was 72, and she was practically symptom free.

Summary and Conclusions

1. Irradiation with either roentgen-rays or radium may be accepted as a useful method of treatment of hyperthyroidism, since the end results are approximately equal to those obtained by surgery.

2. The fear of operation or delays preceding operation are likely to lead to cardiac impairment. This delay can be eliminated by the use of irradiation therapy.

3. Irradiation therapy involves no pain, shock or great inconvenience.

4. Associated medical care and general directions for the conservation of energy are essential.

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THE CAUSE OF DEATH OF PATIENTS WITH ORGANIC HEART DISEASE SUBJECTED TO SURGICAL OPERATION*

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An evaluation of the ability of the patient with organic heart disease to withstand operation is a matter of great importance and also of great difficulty. In 1930, Butler, Feeney and Levine[1] wrote on the cardiac patient as a surgical risk. They reviewed 414 cases with organic heart disease who had been subjected to operation. Their study, contrary to some previous reports, indicated that these patients tolerate operation reasonably well. In their series, comprising 494 operations, there were 60 deaths, a mortality of 12.1 per cent. If those patients whose cardiac condition was such as to have produced death regardless of the operation, or whose surgical condition alone was sufficient to cause death, be excluded the mortality (unexpected mortality) was only 6.3 per cent. The present study consists of a detailed analysis of the causes of death in the 60 cases that were included in their report as compared with 60 non-cardiac patients who died following operation. It is hoped that such a study will throw some light upon the part played by an organically diseased heart in operative and postoperative mortality.

The patients comprising the 60 fatalities, as described in the original report, presented various types of heart disease including chronic valvular disease, chronic adhesive pericarditis, chronic myocarditis, angina pectoris, coronary thrombosis and thyroid heart disease. These cases are briefly summarized in table 1. The second group of non-cardiac cases was chosen from the surgical deaths of the Peter Bent Brigham Hospital regardless of the type of operation or anesthesia, the only essential criteria being that they had no history or physical signs of heart disease and, in the cases where an autopsy was done, showed negligible evidence of organic disease of the heart. An attempt was made to include the older patients in order that the ages in this group might be comparable to those in the cardiac group. Whereas the 60 deaths in the cardiac group resulted from 494 operations on 414 patients, the 60 deaths in the non-cardiac group represent the mortality (3.7 per cent) from approximately 1600 operations. In subsequent analysis it will be important to bear this difference in mind.

Early in the study it became obvious that it is not always easy, even in instances where an autopsy has been done, to state precisely the cause of death. After most careful study there remained in each group a number of cases where the cause of death had to be listed as unknown. This difficulty in deciding upon a single cause of death was more apparent in the

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From the Medical Clinic of the Peter Bent Brigham Hospital, Boston, Massachusetts.

TABLE I
Cardiac Patients
Part A. (Death from Miscellaneous Causes)

Age	Sex	Surgical Diagnosis	Cardiac Diagnosis	Operation	Anesthetic	Course
70	F	Fracture femur	Chronic myocarditis	Application plaster	Gas-oxygen	Temperature rise and bronchopneumonia at both bases. Death 9th day.
48	M	Empyema of pleura	Fibrous pericarditis	Thoracostomy	Novocaine	Continued septic course. Death 8 hours p.o. Autopsy showed lobar pneumonia, empyema of pleura and mediastinum and pericarditis.
44	F	Umbilical hernia	Mitral stenosis	Repair of hernia	Ether	Soon after operation increasing dyspnea, extrasystoles, râles at bases. Temperature rise to 101.2° F. Sudden change and death on 3rd day. Autopsy showed bronchopneumonia.
25	F	Oöphoritis	Endocarditis (acute)	Oöphorectomy	Gas-oxygen	Rapid pulse and delirium, fever, decline and death in 3 months. No positive blood cultures. Cause of death endocarditis (? Gonococcus).
62	F	Varicose ulcer	Chronic myocarditis	Excision skin graft	Spinal novocaine	Continued fever reaching 101° F. on 10th day. Declining fever, increasing stupor. Autopsy not done. Cause of death unknown.
63	M	Cholelithiasis	Chronic myocarditis	Choledochostomy	Ether	Anuria. Pulse became irregular and weak. Terminal temperature rise. Death 2nd day. Autopsy not done. Cause of death unknown.
71	F	Carcinoma of breast	Chronic myocarditis	Amputation of breast	Ether	Soon after operation became disoriented and incontinent. Gradual decline. Cheyne-Stokes respiration and uremia. Cause of death: uremia.
70	F	Gangrene of foot	Chronic myocarditis	Amputation of leg	Gas-oxygen	Increasing stupor, rising temperature and dullness at lung bases. Cause of death: bronchopneumonia.
61	F	Carcinoma of rectum	Chronic myocarditis	Kraske 1. 2.	Ether Gas-oxygen	Nine days after second operation, temperature rise and edema of feet. Infected wound. On 14th day suddenly more dyspneic and died. Cause of death: (?) infection or congestive failure.
78	M	Duodenal obstruction	Chronic myocarditis	Gastro-jejunostomy	Novocaine	On 8th day rise in temperature and signs of consolidation at bases. Death 9th day. Autopsy showed bronchopneumonia and empyema on the right.
69	F	Embolism (popliteal)	Chronic myocarditis	Thigh amputation	Sacral and ether	At operation thrombus found in femoral vein, sudden death 2nd day. Autopsy showed pulmonary embolus.
60	M	Cholecystitis	Chronic myocarditis	Cholecystectomy	Ether	Third day fever and bronchial breathing at left base. Increasing respirations, cyanosis and death. Terminal temperature 102° F. Cause of death: bronchopneumonia.
72	M	Strangulated hernia	Chronic myocarditis	Repair of hernia	Novocaine	Second day marked dyspnea and cyanosis. Increasing coma and death. Cause of death unknown.

Part A. (*Continued*)

Age	Sex	Surgical Diagnosis	Cardiac Diagnosis	Operation	Anesthetic	Course
19	F	Mitral stenosis	Same	Valvulotomy	Ether	Third day rise in temperature and consolidation at left base. Septic course and death on 7th day.
71	M	Prostatism	Chronic myocarditis	Cystostomy Prostatectomy	Novocaine Ether	Failed rapidly on 2nd day after prostatectomy, became dyspneic and cyanotic and died. Autopsy failed to show adequate cause for death. Cause unknown.
76	M	Duodenal obstruction	Chronic myocarditis	Gastroenterostomy	Novocaine	Immediately after operation had Cheyne-Stokes respiration but seemed to improve. Second day became progressively more dyspneic and died. Cause of death unknown.
78	F	Carcinoma pancreas	Chronic myocarditis	Choledochoenterostomy	Novocaine and ether	On 9th day rising pulse, falling blood pressure and death. Autopsy showed massive intestinal hemorrhage from duodenal ulcer.
77	M	Prostatism	Chronic myocarditis	Prostatectomy	Spinal	Rising temperature and death 2nd day. Autopsy showed pelvic and generalized peritonitis and passive congestion of lungs.
62	M	Hyperthyroidism	Thyroid heart	Thyroidectomy	Gas-oxygen	Death 2nd day in thyroid storm.
16	F	Appendicitis (ruptured)	Mitral stenosis	Appendectomy drainage	Ether	Discharged from hospital 27th day. Returned shortly and died of vegetative endocarditis.
56	M	Thrombosis femoral art.	Coronary thrombosis	Leg amputation	Spinal	Sudden death on 2nd day. Autopsy showed pulmonary embolism.
45	F	Exophthalmic goiter	Thyroid heart	Ligation of sup. poles	Gas-oxygen novocaine	Death 8 hours after operation in thyroid storm.
60	F	Cholelithiasis	Chronic myocarditis	Cholecystectomy	Novocaine and ether	Drainage of *B. Coli* pus 31st day. Septic course. Death 41st day. Autopsy showed subphrenic abscess and general sepsis.
68	F	Gangrene foot	Chronic myocarditis	Foot amputation	Spinal	Fall in blood pressure during operation, continued slight fever. Death 4th day. Autopsy failed to show adequate cause for death.
56	F	Intestinal obstruction	Chronic myocarditis	Enterostomy	Gas-oxygen	Fall in blood pressure at 24 hours. Hiccough, rising B.U.N. Terminal temperature rise. Autopsy showed chronic nephritis, infection abdominal wall. Cause of death: uremia.
64	M	Perforated ulcer	Chronic myocarditis	Plication of ulcer. G-E	Gas-oxygen	Rising B.U.N. and death 10th day. Autopsy showed subphrenic abscess. Cause of death: uremia, infection.

Part A. (*Continued*)

Age	Sex	Surgical Diagnosis	Cardiac Diagnosis	Operation	Anesthetic	Course
74	F	Carcinoma; int. obs.	Chronic myocarditis	Colostomy	Novocaine	Falling blood pressure, distention, decline and death 8th day. Autopsy showed carcinomatosis, bronchopneumonia, hydrothorax. Cause of death: cachexia, bronchopneumonia.
54	M	Carcinoma of stomach; cholelithiasis	Chronic myocarditis	Gastro-jejun. Cholecystectomy	Novocaine and ether	Fifth day respirations increased. Seventh day roentgen-ray showed bronchopneumonia. Decline and death. Autopsy showed bronchopneumonia and carcinomatosis.
59	M	Hernia	Chronic myocarditis	Repair of hernia	Novocaine	Increasing uremia and death 13th day.
60	F	Procidentia	Chronic myocarditis	Ventral fixation	Ether	Sudden death on 16th day. Cause of death: pulmonary embolus.
72	F	Carcinoma bile ducts	Coronary sclerosis	Cholecystectomy	Gas-oxygen ether	Continued state of shock, lapsed into coma and died on 3rd day. Cause of death: cachexia and bronchopneumonia.
43	M	Intestinal obstruction	Hypertension Myocarditis	Release bands Enterostomy	Novocaine	Death on 8th day of peritonitis and partial obstruction.
48	F	Cholelithiasis	Chronic myocarditis	Cholecystectomy	Spinal, gas and ether	Death on 2nd day from liver necrosis due to accidental ligation hepatic artery.
66	M	Prostatism; empyema	Chronic myocarditis	Prostatectomy; thoracostomy	Gas-oxygen Novocaine	Eighteenth day after operation developed empyema. Later phlebitis. Long septic course and final death from pulmonary embolus.
21	F	Mitral stenosis	Same	Valvulotomy	Ether	Second day death with lobar consolidation at both bases. Confirmed by autopsy.
32	M	Abscess of thigh	Mitral and aortic stenosis	Incision and drain 1, 2, 3, 4	Gas-oxygen	Sudden death apparently from pulmonary embolism.
73	M	Gangrene of of toe	Chronic myocarditis	Amputation	Novocaine	Spreading infection in foot and leg. Death from bronchopneumonia 19th day.
61	F	Strangulated hernia	Chronic myocarditis	Repair of hernia	Novocaine	Sudden death 4 hours after operation, interpreted as pulmonary embolism.

Part A. (*Continued*)

Age	Sex	Surgical Diagnosis	Cardiac Diagnosis	Operation	Anesthetic	Course
58	M	Carcinoma stomach	Endocarditis (acute)	Laparotomy	Ether	Up in chair 29th day. Slight fever. Pulmonary infarcts. Hemiplegia, decline and death 47th day. Autopsy: thromboses (inferior vena cava, pulmonary arteries, coronary), acute endocarditis and carcinoma.
66	F	Carcinoma sigmoid	Chronic myocarditis	Sigmoid-ostomy	Novocaine	Incarcerated diaphragmatic hernia not relieved at operation. Decline and death 12 hours after operation. Autopsy: cause of death, intestinal obstruction.
67	M	Prolapse rectum	Chronic myocarditis	Whitehead (modified)	Novocaine	Increasing stupor, rising B.U.N. Death from uremia (autopsy).
49	F	Carbuncles	Chronic myocarditis	Incision and drainage	Gas-oxygen	Continued stupor, decline and death. Cause of death unknown.
65	M	Carcinoma penis	Chronic myocarditis	Resection of glans	Novocaine	Bronchopneumonia 9th day. Decline and death. Autopsy showed thrombosed iliac veins, bilateral pulmonary emboli with congestion and edema. Cause of death: pulmonary embolism.
64	F	Carcinoma esophagus	Chronic myocarditis	Gastrostomy	Novocaine	Decline and death on 23rd day. Autopsy showed mediastinal abscess and bronchopneumonia.
65	M	Carcinoma bladder	Coronary sclerosis	Excision of tumor	Spinal	Sudden death 27th day. Autopsy showed pulmonary embolus.
				Part B. (Death from Congestive Heart Failure)		
37	F	Valvular D. Ad. pericard.	Same	Cardiolysis	Ether	Immediately after operation began to fill with edema and died in 8 hours. Autopsy: mitral stenosis, tricuspid stenosis, old infarcts in lungs, etc.
43	M	Adhesive pericarditis	Same	Cardiolysis	Ether	Operation lasted 4 hours. Lungs soon filled with fluid, increasing dyspnea and death on 3rd day. Autopsy limited to heart.
72	M	Prostatism	Chronic myocarditis	Prostatectomy	Spinal	On 2nd day developed auricular fibrillation, increasing dyspnea and died. Autopsy showed interlobar effusion, slight bronchopneumonia.
35	F	Mitral stenosis	Same	Cardiotomy Valvulotomy	Ether	Long operation. Heart stopped once during procedure. P.O. increasing dyspnea and death within 24 hours. Autopsy limited to the heart.
63	M	Cholelithiasis	Chronic myocarditis	Cholecystectomy	Ether	Second day developed moist râles at bases. Digitalized without effect and died 2nd day. Autopsy showed hydrothorax, pulmonary congestion and early bronchopneumonia.

Part C. (Death Due to Coronary Occlusion)

Age	Sex	Surgical Diagnosis	Cardiac Diagnosis	Operation	Anesthetic	Course
64	M	Prostatism	Cardiac infarct	Prostatectomy	Spinal	Wound infection. Attempted secondary closure 27th day. 29th day suddenly dyspneic, cyanotic, died in 30 minutes. Autopsy: large softening in heart, mural thrombi, congestion.
55	F	Empyema	Coronary occlusion	Thoracostomy	Novocaine	Developed empyema during treatment for coronary occlusion. Seventh day after operation suddenly more dyspneic, cyanotic and died.
68	F	Cystocele rectocele	Chronic myocarditis	Hysterectomy Repair	Ether	First carried to operating room, had a fall in blood pressure and operation was delayed 2 days. Immediately after operation again had fall in blood pressure; 9th day suddenly became weak and died.
51	M	Angina pectoris	Syphilis of heart	Cervical sympathect.	Ether	Sudden death 6 hours after operation. Autopsy showed occlusion of right coronary.
70	M	Carcinoma bladder Prostatism	Cardiac infarct	Excision and prostatectomy	Gas-oxygen	Sudden death on 3rd day. Autopsy showed cardiac infarct with rupture.
56	M	Thrombosis femoral art.	Angina pectoris	Leg amputation	Novocaine and spinal	Coronary occlusion 1 week previously. Hurried amputation. Decline and death 2nd day. Autopsy showed extensive cardiac infarct and many small pulmonary emboli.
42	M	Gangrene toe	Coronary occlusion	Popliteal ligation Amputation	Novocaine Gas-oxygen	Ten days after amputation was apparently doing well when he suddenly became dyspneic and died. Autopsy showed thrombosis left coronary, mural thrombi in aorta and femoral artery.
58	F	Hemorrhoids	Cardiac infarct	Hemorrhoidectomy	Gas-oxygen	Operation immediately followed by dyspnea and cyanosis. Electrocardiogram showed coronary changes. Flaccid paralysis on left, rapid decline and death on 4th day. Autopsy showed large cardiac infarct. Also cerebral infarct.

Part D. (Death Due to Cerebral Accidents)

82	M	Osteomyelitis of toe	Chronic myocarditis	Amputation of toe	Gas-oxygen	Uneventful course until 7th day when suddenly became unconscious, developed hemiplegia, tracheal râles, and died.
55	M	Aneurysm (ulnar)	Mitral stenosis	Ligation brachial	Ether	Satisfactory course until 4th day, suddenly unconscious, developed hemiplegia, progressive decline and death on 22nd day.

cardiac group. This difference is probably, in a measure at least, due to a greater occurrence in the cardiac group of such more or less intangible factors as peripheral vascular collapse. In such instances one cannot attribute the cause of death to the heart per se though the damaged heart may contribute to the causation or continuation of such a state of vascular collapse. This state of impaired circulation probably also predisposes to uremia in instances where the kidney function has previously been impaired and also predisposes to thromboses in the visceral, cardiac, and cerebral vessels if these structures are the site of any preëxisting disease.

TABLE II

	Cardiacs	Non-Cardiacs
Number of patients	414	1600
Number of deaths	60	60
Autopsies	38	43
Average age	58.9 years	51.5 years
Males	30	39
Females	30	21

TABLE III

Causes of Death

	Cardiacs		Non-Cardiacs	
	Chief	Contributing	Chief	Contributing
Unknown	9		6	
Congestive heart failure	5	9	0	0
Coronary occlusion	8	1	0	0
Cerebral accident	2	3	0	0
Pulmonary embolus	7	2	10	1
Infection	17	16	29	8
Infarction	0	7	1	2
Uremia	5	0	2	1
Hemorrhage	1	0	3	0
Cachexia	1	3	4	7
Thyroid storm	3	0	0	0
Liver necrosis	1	0	0	0
Intestinal obstruction	1	0	0	0
Purpura	0	0	1	0
Air embolus	0	0	1	0
Anesthetic	0	0	2	0
Hyperinsulinism	0	0	1	0

Comparative data on the two groups are presented in tables 2 and 3. In the group of cardiac patients there were 30 males and 30 females with an average age of 58.9 years. Thirty-eight of these were studied post mortem. In the group of non-cardiac patients there were 39 males and 21 females with an average age of 51.5 years. Forty-three autopsies were performed in this group. The younger age in the latter group is undoubtedly due to the fact that many patients past 50 years of age had either clinical or pathologic evidence of some degree of heart disease and therefore had to be excluded. Analysis of the causes of death shows several things of note. In the cardiac group there are causes such as congestive heart failure, coronary occlusion and cerebral accidents which do not exist in the non-cardiac as principal or contributing causes, the difference being

largely made up by the greater incidence of infection in the latter group. There is also a much greater incidence of contributing causes in the cardiac group. Contrary to what one might have expected, infarction other than that resulting from coronary occlusion, though frequently a contributing cause, was not chiefly responsible for death in any patient of group one. Pulmonary embolism appears more frequently as a cause of death in the non-cardiac group. This does not by any means indicate that pulmonary embolism occurs more frequently in the non-cardiac patient. Indeed it must be apparent that if pulmonary embolism occurred seven times as a cause of death in 414 patients and only 10 times as a cause of death in 1600 non-cardiac patients, the true incidence is considerably higher in the former group. By a similar analysis the incidence of fatal hemorrhage in the two types of cases appears to be about the same. In this study the unusual causes of death do not occur with sufficient frequency to warrant comparison of the two groups. The greater incidence of uremia in the cardiac patients is to be expected because of the association of renal and cardiac disease.

Infection was the most common cause of death in both groups. A study of the location of infection indicates three chief sites: (1) lung and pleura, (2) peritoneum, (3) generalized infection (septicemia). In table 4, it is seen that the incidence of the former and latter is the same in the cardiac and non-cardiac patients. Let us again point out here the difference between the composition of the two groups. If fatal pulmonary infection occurred 11 times in 414 cardiacs and only 11 times in 1600 non-cardiacs, the liability to pulmonary infection is greater in the former group. The same analysis holds for generalized infection. The greater incidence of peritoneal infection in the non-cardiac group is probably due to the fact that in this group, which is lacking in certain causes of death such as coronary occlusion, in order to gather 60 deaths it was necessary to include a greater number of patients with peritonitis. This difference in incidence does not represent the result of a variation in immunity or susceptibility in the two groups. It is entirely probable that, given an equal number of operations for acute appendicitis or perforated ulcer in cardiacs and non-cardiacs, the incidence of fatal peritonitis would be equally high in the two types of cases.

TABLE IV

Site of Fatal Infection

	Cardiac	Non-cardiac
Lung and pleura	11*	11
Peritoneum	1	14
General	3	3
Pericardium	1	0
Mediastinum	1	0
Liver	0	1

* In the cardiac patients pulmonary infection was frequently associated with pulmonary congestion or infarction.

Let us now consider those causes such as congestive heart failure, coronary occlusion, and cerebral accidents which do not exist in the non-cardiac cases (see table 1, parts B, C, and D). Such causes accounted for one-fourth of the deaths in the cardiac patients. Of the five instances of congestive heart failure as a cause of death three were patients who had operations on the heart itself (cardiolysis or valvulotomy) and may therefore be disregarded insofar as general operative mortality is concerned. There remain then only two cases where congestive failure was the major factor in causing death. In view of the fact that a large number of the patients comprising the original series of 414 patients had evidence of congestive failure at some time during the period of observation this low percentage appears definitely to indicate that congestive heart failure is not a major hazard in operations upon the well treated cardiac. Its effect as a contributing cause of death is difficult to assess. Along with infarction it was probably a factor in causing the relatively greater incidence of fatal pulmonary infection in the cardiac group. Often in the cases where pulmonary infection was decided upon as the cause of death there was also clinical or pathologic evidence of pulmonary congestion or infarction. Wherever it seemed probable that in the absence of infection the individual might have survived the effects of the other pulmonary pathology the cause of death has been attributed to the infection. When we come to consider the cases where coronary occlusion was the cause of death it is clear that, though some of this group of eight patients would undoubtedly have died even if the operation had not been done, there were a number who might otherwise have survived. Cerebral accident appears to have been the cause of death in only two instances though its incidence as a contributing factor is slightly greater. It is probably true that in the cardiac the liability to cerebral accidents is somewhat increased by operation.

What causes, then, account for the increased hazard of operations upon the cardiac patient as compared with the non-cardiac patient? The present study indicates that congestive heart failure is not a significant factor in increasing the mortality of the cardiac except as it may contribute to a predisposition to pulmonary infection. The cardiac patient in a ratio of about four to one seems more liable to fatal pulmonary infection than does the non-cardiac. The greater incidence of fatal pulmonary embolism and the effect of coronary occlusion or its sequellae are likewise major factors in increasing the risk of operation upon the cardiac patient.

Summary

(1) The causes of death of 60 patients with organic heart disease who have been subjected to operation are compared with the causes of death in a similar group of 60 non-cardiac patients.

(2) This study indicates that in the well treated cardiac, congestive

heart failure is not a very significant factor in the causation of death following operation.

(3) The cardiac group differs from the non-cardiac chiefly in the presence of fatal coronary occlusion and in the greater incidence of fatal pulmonary complications, chiefly infection and pulmonary embolism.

I wish to express my appreciation to Dr. Samuel A. Levine for his assistance in this study.

REFERENCE

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MYELOID INSUFFICIENCY *

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There is a group of anemias with a blood picture of an exhausted myeloid system, showing little or no signs of regeneration. The blood picture is that of an anemia, granulocytopenia, and thrombocytopenia. In some of these cases the etiology is known, e.g., arsenic or benzol poisoning, in others the etiological factor cannot be found. In the literature these cases are referred to as " aplastic anemia," " myelophthisic anemia," " aleukia hemorrhagica," etc., but none of these terms properly expresses the character of the condition. We are really dealing with an insufficiency of the myeloid system. A myeloid insufficiency may involve the entire system or only parts of it. In the so-called " malignant neutropenia " or " agranulocytosis " only the granulopoietic apparatus is involved. The close pathogenic relationship of these cases of agranulocytosis and aplastic anemia is expressed in the fact that a number of the agranulocytoses eventually develop the picture of a complete myeloid insufficiency with anemia and thrombocytopenia.[1] The same is true of arsenic and of benzol poisoning in which either part or the whole myeloid system may be involved. The question now arises as to the way in which a poison or toxin affects the myeloid system. Is it a direct action on the myeloid system, or does the toxin or poison act through a disturbance of the normal balance in the hematopoietic system? It is a well known fact that the normal myeloid system per se has a tendency to extensive proliferation; this proliferation is kept in check by the reticulo-endothelial system. A removal of the normal inhibition leads to an extensive proliferation of the myeloid system, as can be seen experimentally in animals after the removal of the spleen, or in the human after a normal spleen has been removed for some mechanical reason (e.g., traumatic ruptured spleen). The myeloid proliferation lasts as long as it takes for the reticulo-endothelial system to make up for its loss by proliferation in other places (Kupffer cells, accessory spleens, etc.). We know that the normal reticulo-endothelial system has an inhibitory effect on the normal myeloid system. In infections in which a reticulo-endothelial proliferation is met with, the myeloid system is usually depressed. Such is the case for instance in typhoid fever, influenza, miliary tuberculosis and in other conditions in which leukopenia, anemia, and purpura are so frequently present. Although a direct action of an infectious agent, a poison or a toxin, on the myeloid system cannot entirely be denied, or at least not at certain stages, the importance of a disturbed equilibrium of hematopoiesis must be borne in mind, especially the possibility that a stimulated and pro-

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liferated reticulo-endothelial system may increase its physiological inhibition to a pathological degree. If such a pathological inhibition persists even after the original cause has been removed, it is only logical that a complete atrophy of the myeloid system may result.

The case presented here is an example of a complete myeloid insufficiency of unknown etiology.

Case Report

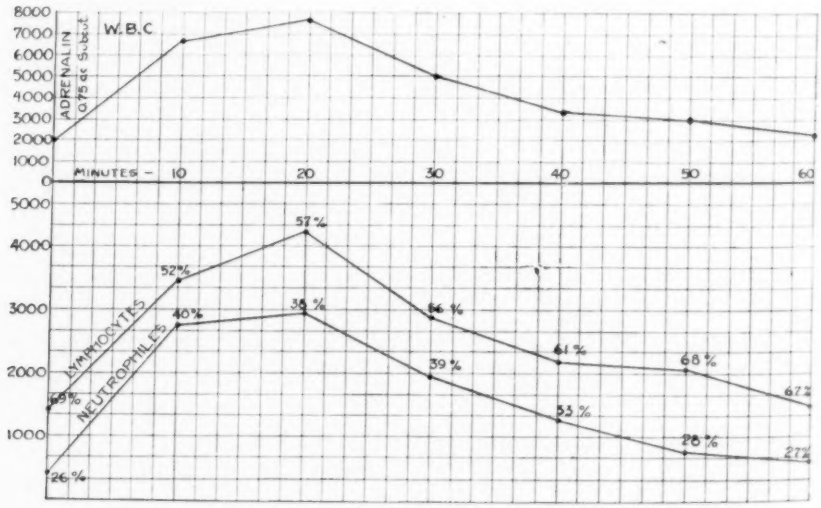
A male patient, age 19, first came under observation on August 1, 1932. His only complaints were progressive weakness, pallor, and bleeding from the gums. His past history was of no importance. He was a high-school student and very active in athletics. About one year previously, the patient had noticed he would tire more quickly when playing tennis or other games but, being otherwise well, he ignored this. In April 1932 the patient had a cold which lasted for about two weeks; at this time he first noticed bleeding from his gums. Since that time he became progressively weaker and paler, and bleeding from the gums became quite profuse during July. On July 20, he received a transfusion, with improvement for a few days.

On August 1, 1932, examination revealed the following facts: temperature normal, pulse 96, respiration 20, good nutrition, marked pallor with a slightly greenish tinge, bleeding from the gums, positive Frank-Hess sign, spleen just palpable. The physical examination did not otherwise reveal anything abnormal. Urine, stool, and gastric analyses were normal.

Hematological examination: Red blood cells 1,270,000; hemoglobin 32 per cent (4.48 gm./100 c.c. of blood); color index 1.3; white blood cells 2,550; thrombocytes 68,000; reticulocytes 2 per cent. The differential count showed a marked granulocytopenia and 69 per cent lymphocytes. Bleeding time was prolonged; coagulation time normal; clot retraction absent.

In spite of the normal gastric acidity 20 c.c. of liver extract (P.D.) were injected intravenously on the same day without any appreciable effect for the next 10 days.

Chart 1. Adrenalin test (August 5, 1932)



The patient was admitted to the hospital on August 5, his condition having become worse. Examination showed: Red blood cells 1,120,000; hemoglobin 25 per cent; color index 1.13; white blood cells 2,150; thrombocytes 10,000. Profuse bleeding from the gums was present. The adrenalin test showed a fair myeloid response in 20 minutes, leading to an increase of the white blood cells from 1,900 to 7,800, with a return to the original level within one hour. (See chart 1.)

The clinical picture, together with the hematological examination, suggested an exhausted myeloid system, leading to a reduction of all myeloid elements, that is, anemia, granulocytopenia, and thrombocytopenia; signs of regeneration, although not entirely absent, were slight.

Based on the assumption of the reticulo-endothelial inhibition being the cause of the myeloid insufficiency it was decided to remove the spleen in order to relieve the myeloid system of part of that inhibition. The absence of an infection and the fair myeloid response in the adrenalin test were further aids in the decision.

Two transfusions were given to the patient prior to the operation, and the spleen was removed on August 10. The spleen was slightly larger than normal. Sections showed a thin overlying capsule, normal appearing trabeculae, well formed vascular channels free from hyaline changes and surrounded by a comparatively large amount of lymphoid tissue for this age period. The pulp was cellular, containing in addition to numerous lymphocytes occasional polymorphonuclear and eosinophile leukocytes. The reticular cells were hyperplastic and appeared as large phagocytic elements. The venous sinuses were not conspicuous and appeared rather compressed by the pulp and were lined by rather swollen looking reticulo-endothelium which projected into

TABLE I

Detail blood findings

Date—1932	August 1,	August 5	August 6	August 7	August 8
Hb.	32%	25%	35%	45%	45%
	4.48 gm.	3.25 gm.	4.90 gm.	6.30 gm.	6.30 gm.
R.B.C	1,270,000	1,120,000	1,630,000	1,800,000	1,700,000
C.I	1.33	1.13	1.09	1.25	1.32
Reticulocytes	2%	2½%	2½%	2%	2%
Nucleated R.B.C	0	0	0	0	0
W.B.C	2550	2150	1800	1900	2000
Basophiles	0	0	0	0	0
	0	0	0	0	0
Eosinophiles	3%	2%	2%	3%	3%
	75	42	36	57	60
Myeloblasts	0	0	0	0	0
	0	0	0	0	0
Myelocytes	0	0	0	0	0
	0	0	0	0	0
Meta. myelocytes	0	0	0	0	0
	0	0	0	0	0
Stab. forms	1%	2%	3%	4%	4%
	25	42	54	76	80
Polysegmented	25%	24%	25%	23%	25%
	625	504	450	247	500
Lymphocytes	69%	69%	68%	67%	65%
	1725	1449	1224	1273	1300
Monocytes	2%	3%	2%	3%	3%
	50	63	36	57	60
Thrombocytes	68,000	10,000	45,000	40,000	40,000
Remarks	6 days after transfusion	Admission	24 hrs. after transfusion		

TABLE I (*continued*)

Date	August 9	August 10 9 a.m.	August 10 ½ hour P.O.	August 10 4 hours P.O.	August 11
Hb	41%	46%	44%	44%	42%
	5.74 gm.	6.44 gm.	6.16 gm.	6.16 gm.	5.88 gm.
R.B.C	1,900,000	2,100,000	2,100,000	2,000,000	2,000,000
C.I	1.07	1.09	1.04	1.1	1.05
Reticulocytes	2%	2%	2%	2%	2½%
Nucleated R.B.C	0	0	0	0	0
W.B.C	2200	1900	3100	3100	3000
Basophiles	0	0	0	0	0
	0	0	0	0	0
Eosinophiles	2%	2%	0	0	0
	44	38	0	0	0
Myeloblasts	0	0	0	0	1%
	0	0	0	0	30
Myelocytes	0	0	2%	3%	5%
	0	0	62	93	150
Meta. myelocytes	0	0	3%	5%	7%
	0	0	93	155	210
Stab. forms	3%	3%	5%	22%	15%
	66	57	155	682	450
Polysegmented	26%	23%	22%	20%	17%
	575	247	682	620	510
Lymphocytes	67%	70%	64%	45%	52%
	1474	1330	1984	1395	1560
Monocytes	2%	2%	4%	5%	3%
	44	38	124	155	90
Thrombocytes	30,000	55,000	150,000	250,000	175,000
Remarks	Transfusion 500 c.c.	Splenectomy	½ hour P.O.	4 hours P.O.	

TABLE I (*continued*)

Date	August 12	August 13	August 15	August 16	August 17
Hb	45%	45%	48%	50%	48%
	6.30 gm.	6.30 gm.	6.72 gm.	7.00 gm.	6.72 gm.
R.B.C	2,200,000	2,310,000	2,420,000	2,575,000	2,630,000
C.I	1.02	0.97	1	1	0.92
Reticulocytes	4½%	5%	5½%	6%	5%
Nucleated R.B.C	+	+	++	+	+
W.B.C	3100	3300	3600	3900	3950
Basophiles	0	0	0	0	1%
	0	0	0	0	39
Eosinophiles	0	0	1%	2%	2%
	0	0	36	78	78
Myeloblasts	2%	2%	2%	1%	0
	62	66	72	39	0
Myelocytes	5%	4%	5%	4%	3%
	155	132	180	156	117
Meta. myelocytes	9%	6%	5%	5%	4%
	279	198	180	195	156
Stab. forms	16%	12%	11%	10%	10%
	496	396	396	390	390
Polysegmented	13%	20%	20%	22%	24%
	403	660	720	858	936
Lymphocytes	51%	52%	52%	53%	54%
	1581	1716	1872	2067	2106
Monocytes	4%	4%	4%	3%	3%
	124	132	144	117	117
Thrombocytes	125,000	100,000	125,000	137,000	122,000
Remarks					

TABLE I (*continued*)

Date	August 19	August 22	August 24	August 26	August 29	September 1
Hb	45% 6.30 gm.	46% 6.44 gm.	45% 6.30 gm.	40% 5.60 gm.	42% 5.88 gm.	44% 6.16 gm.
R.B.C	2,450,000	2,540,000	2,400,000	2,200,000	2,250,000	2,400,000
C.I	0.93	0.92	0.93	0.90	0.95	0.91
Reticulocytes	5%	5%	8%	7.5%	7%	5.5%
Nucleated R.B.C	0	0	0	0	0	0
W.B.C	4100	4200	4600	4900	5900	5800
Basophiles	0	0	0	0	0	0
	0	0	0	0	0	0
Eosinophiles	2%	2%	2%	2%	2%	2%
	82	84	92	98	118	116
Myeloblasts	2%	1%	0	1%	0	0
	82	42	0	49	0	0
Myelocytes	4%	2%	3%	2%	2%	1%
	164	84	138	98	118	58
Meta. myelocytes	4%	2%	2%	3%	4%	5%
	164	84	92	147	236	290
Stab. forms	10%	10%	8%	10%	12%	12%
	410	420	368	490	708	696
Polysegmented	25%	34%	36%	38%	36%	37%
	1025	1428	1656	1862	2124	2146
Lymphocytes	50%	47%	47%	41%	41%	40%
	2050	1974	2162	2009	2419	2320
Monocytes	3%	2%	2%	3%	3%	3%
	123	84	92	147	117	174
Thrombocytes	133,000	142,000	122,000	100,000	150,000	148,000
Remarks						

TABLE I (*continued*)

Date	September 10	September 19	September 26	October 1	October 6
Hb	70% 9.80 gm.	82% 11.48 gm.	48% 6.72 gm.	60% 8.40 gm.	60% 8.40 gm.
R.B.C	3,600,000	4,300,000	2,700,000	3,180,000	3,220,000
C.I	0.97	0.95	0.88	0.96	0.93
Reticulocytes	4%	4%	12%	6%	2%
Nucleated R.B.C	0	0	++	+	0
W.B.C	6300	6400	7200	6200	7100
Basophiles	0	0	0	0	0
	0	0	0	0	0
Eosinophiles	2%	2%	2%	3%	3%
	126	128	144	186	213
Myeloblasts	0	0	0	0	0
	0	0	0	0	0
Myelocytes	0	0	3%	0	0
	0	0	216	0	0
Meta. myelocytes	2%	2%	6%	3%	3%
	126	128	432	186	213
Stab. forms	8%	6%	15%	10%	11%
	504	374	1080	620	781
Polysegmented	44%	45%	30%	40%	39%
	2772	2880	2160	2480	2769
Lymphocytes	39%	40%	38%	40%	40%
	2457	2560	2736	2480	2840
Monocytes	5%	4%	6%	4%	4%
	305	256	432	248	284
Thrombocytes	162,000	146,000	79,000	153,000	170,000
Remarks			Cold. Transfusion 500 c.c.		

TABLE I (*continued*)

Date	October 10	October 15	October 18	October 20	October 24
Hb	50% 7.00 gm.	35% 4.90 gm.	35% 4.90 gm.	18% 2.52 gm.	10% 1.40 gm.
R.B.C	2,700,000	1,900,000	1,800,000	910,000	560,000
C.I.	0.92	0.92	0.97	1	1
Reticulocytes	0.5%	0.5%	0	0	0
Nucleated R.B.C	0	0	0	0	0
W.B.C	6800	10,000	12,000	11,000	12,000
Basophiles	0 0	0 0	0 0	0 0	0 0
Eosinophiles	2% 136	2% 200	1% 120	1% 110	0 0
Myeloblasts	0 0	0 0	0 0	0 0	1% 120
Myelocytes	0 0	2% 200	3% 360	2% 220	4% 480
Meta. myelocytes	4% 272	6% 600	6% 720	7% 770	8% 960
Stab. forms	12% 816	14% 1400	12% 1440	13% 1430	15% 1800
Polysegmented	40% 2720	38% 3800	40% 4800	38% 4180	34% 4080
Lymphocytes	37% 2516	32% 3200	33% 3960	34% 3740	32% 3840
Monocytes	5% 340	6% 600	5% 600	5% 550	6% 720
Thrombocytes	120,000	60,000	50,000	20,000	1000
Remarks		Transfusion 500 c.c.	Profuse bleeding	Broncho-pneumonia	

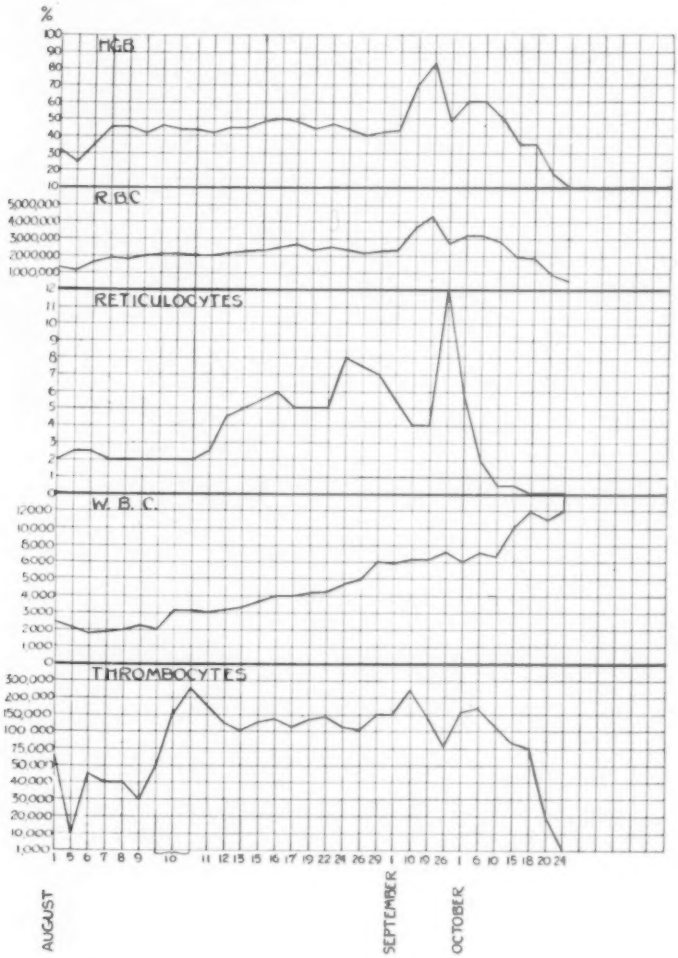
the sinus. Special stains showed the following: (1) There was a moderate number of blue staining cells by the oxydase reaction. These were for the most part found in the cellular exudate in the pulp but there appeared to be occasional small foci of myeloid metaplasia. (2) Stain for reticulin: This was found well preserved as a finely meshed interlacing network. (3) Special stain for collagen fibrils showed no appreciable increase.

Following the splenectomy there was an almost instantaneous improvement. The blood picture quickly assumed a hyperregenerative character (details may be seen in table 1 and chart 2). Figure 1, which is composed from six different fields, shows the different young forms, and the rapidly forming platelets which were often found to be still in protoplasmic connection. The wound healed normally; the patient improved steadily, and was discharged from the hospital on September 2, 1932. From September 2 to September 20 the patient was perfectly well. On September 20 he developed a cold and started to go rapidly downhill. A transfusion was given on September 26 which led to a short temporary improvement. The anemia became progressively worse, however, and a transfusion was again given on October 10 with little success. On October 20 the patient developed bronchopneumonia and died on October 26. An autopsy could not be obtained.

Although the result of the splenectomy showed only a temporary improvement, we feel inclined to believe that our method of procedure was justified. Since patients with acute idiopathic myeloid insufficiency are doomed to die in a short time, any attempt to save them, or at least prolong life, is justified. We may perhaps add that early operation is essential and one should be guided in the indication by the findings of a bone-marrow bi-

opsy (which unfortunately was not done in this case), since one can hardly expect a regeneration to occur from a completely fatty myeloid system. Other points of importance which one must consider are the results of the adrenalin test, and the absence of infection.

CHART 2. Blood findings



SUMMARY

1. A case of idiopathic (essential) myeloid insufficiency is reported.
2. A possible explanation for the pathogenesis is offered.
3. Early splenectomy is suggested as a possible means to combat the disease.

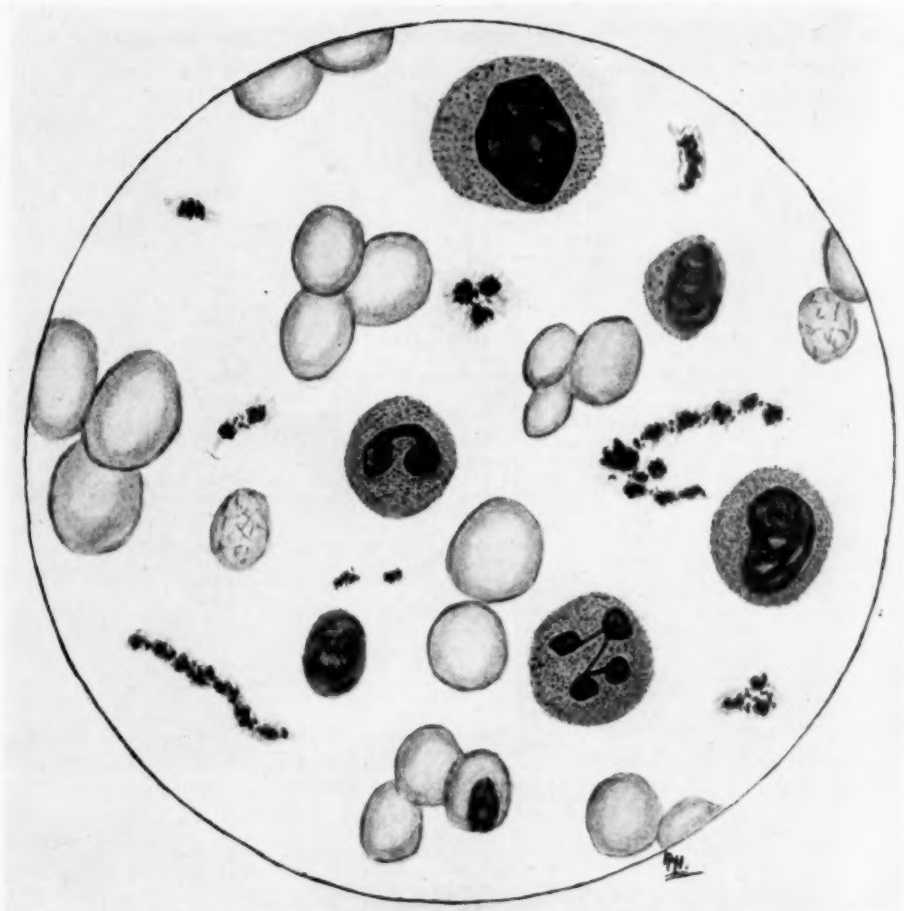


FIG. 1. Blood picture after splenectomy.

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THE IMPORTANCE OF BRONCHOSCOPY IN BRONCHIECTASIS *

By Horace P. Marvin, M.D.,† *Honolulu, Hawaii*

Bronchoscopy plays a very important part in both the diagnosis and treatment of cases of bronchiectasis. Practically all of the limited literature on this subject has been contributed by the bronchoscopist or the surgeon, with an occasional brief note by an internist. Jackson states that " in most cases of bronchiectasis there are strong indications for a bronchoscopic diagnosis." Ballon, Singer and Graham report that " the bronchoscope is a great aid in the diagnosis of bronchiectasis," and also state that " the value of the bronchoscope in the treatment of foreign body bronchiectasis and in certain cases which are due to partial bronchial stenosis is obvious."

It is not the purpose of this paper to attempt any discussion of bronchoscopic technic, but to present the writer's conclusions on the present subject strictly from the standpoint of the internist. These conclusions have been reached after treating 117 cases of bronchiectasis in the past three years, as well as reviewing the clinical records of 83 additional cases which had been treated in this hospital prior to July 1930, making a total of 200 cases. Seventy per cent, or 147, of these 200 cases had diagnostic bronchoscopies, whereas 44 per cent, or 89 patients, had therapeutic bronchoscopies which varied in number from 1 to 63. Ninety per cent of the cases treated in the past three years have been bronchoscoped for diagnostic purposes and approximately 50 per cent received bronchoscopic treatments.

There is no doubt as to the additional diagnostic data which may be obtained from careful bronchoscopy, especially when there is close cooperation between the bronchoscopist and the internist. A careful examination and detailed report by the bronchoscopist may be the deciding factors in determining the exact etiology, the location of the lesions and the diagnosis in a given case. As mentioned above, bronchiectasis caused by foreign bodies or bronchial strictures are readily detected by bronchoscopic examination and the foreign body can usually be removed or the stricture dilated, thus eliminating the cause and making the prognosis much better. Of much importance to the internist is a report which includes the color and condition of the mucous membrane of the bronchi as well as the nature, amount and location of the secretion when present. In the early or milder cases the bronchoscopist usually reports the mucous membrane as grayish and glazed in appearance, with varying amounts of muco-purulent or purulent sputum in the bronchi affected. In the more advanced cases, the mucous membrane is often reported as thickened, leathery or edematous in

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appearance with from 20 c.c. to 60 c.c. of purulent secretion aspirated from the involved areas of bronchiectasis. It is our custom to obtain a smear and culture of the uncontaminated secretion, which is aspirated and sent to the laboratory in a sterile test tube. A number of different organisms have been cultured from the deep sputum thus obtained, but the organisms usually reported by the laboratory as predominant have been a non-hemolytic streptococcus, the *Staphylococcus albus,* Friedländer's bacillus, and the micrococcus catarrhalis. Sometimes the bronchoscopist reports that he can see the dilatations in the terminal bronchi. Not infrequently the retained secretion in the dilated bronchi is so thick and crusted as to require softening by injecting some warm fluid before complete aspiration can be accomplished. This is a very important procedure in such cases prior to injecting lipiodol for bronchography, otherwise the retained crusted secretion will not allow the lipiodol to fill the bronchiectatic cavities, which results in a negative bronchogram.

In addition to the above mentioned diagnostic information gained by bronchoscopy, this procedure allows the operator to inject the lipiodol by means of the bronchoscope. By so doing, the operator knows that the lipiodol is being instilled into the bronchi rather than entering the esophagus. This latter sometimes happens, especially with an unintelligent or uncooperative patient when the lipiodol is instilled without the scope, even when the best technic is used by the operator. We have obtained several excellent bronchograms by using what we term the "instillation method" (without bronchoscopy), but in many cases in 1930 and 1931, we found it necessary to do a bronchoscopic aspiration and injection of lipiodol after attempting the "instillation method" first. Although the patient may attempt complete emptying of his bronchiectatic cavities by careful postural drainage, yet this may not be possible or may be unsatisfactory due to factors already mentioned. At the present time we request a diagnostic bronchoscopy and instillation of lipiodol for bronchograms in all cases. The information gained from the bronchoscopy plus the bronchogram has usually resulted in arriving at an early and satisfactory conclusion as to the exact nature of the pathologic lesions present. These data furnish additional evidence to support the impressions gained from the history, physical examination and the type of sputum noted following postural drainage. In other words, diagnostic bronchoscopy and bronchography either establish the diagnosis early and conclusively or are of material aid in substantiating the diagnosis of bronchiectasis made clinically at the time the patient was admitted to the hospital.

As important as bronchoscopy has proved to be as an aid in the diagnosis of bronchiectasis, yet it is of much more importance from a therapeutic standpoint. It is a generally accepted fact that the treatment of this disease may be summed up in the two words "bronchial drainage." These two words do not cover all types of therapy, but they do express the main objective in our treatment of bronchiectasis. Bronchial drainage may be

obtained in three ways: (a) postural drainage; (b) bronchoscopic drainage; (c) surgical drainage.[1] Postural drainage is by far the most important of the three methods. This should be practiced consistently and energetically and may be accomplished in any one or more of several ways.

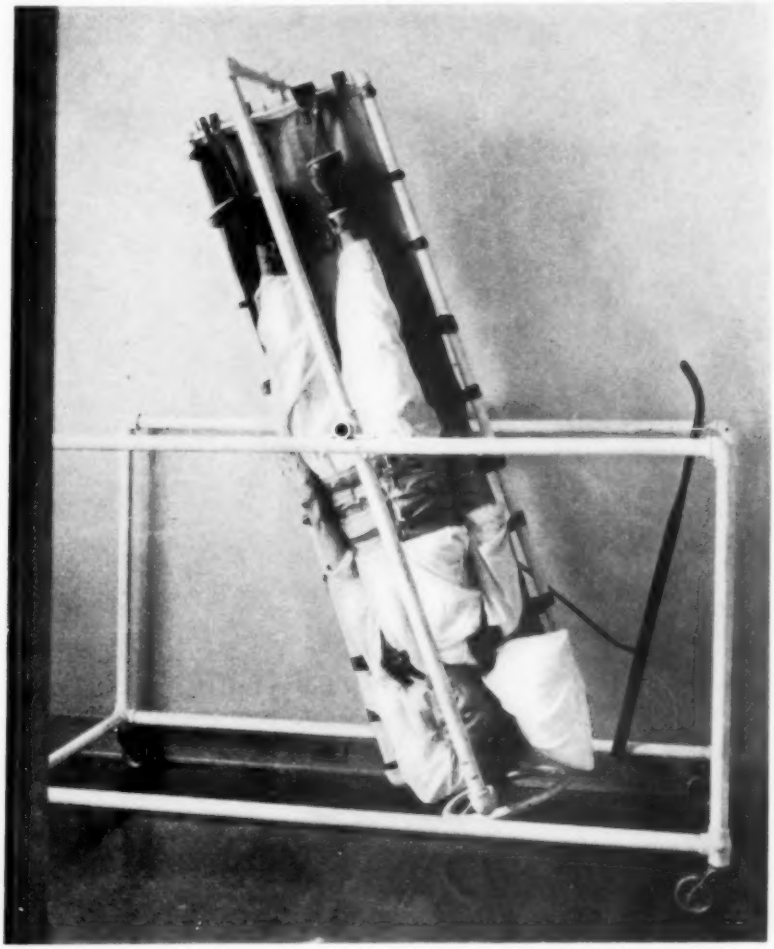

FIG. 1. Adjustable table drainage apparatus. This apparatus allows patient's head to be lowered to any position, as well as side to side adjustment, affording excellent bronchial drainage.

In the milder or moderately severe cases, postural drainage will usually suffice to completely empty the bronchial dilatations and prevent the disease from progressing in its severity or complications from developing. On our bronchiectatic service at this hospital we use a so-called "adjustable table drainage apparatus" (figure 1) and a "Jackknife drain" (figure 2), both of which are very useful and efficient in postural drainage. For the

past four months we have used a Singer multiposition bed which has been very useful in obtaining postural drainage in both pulmonary abscess and bronchiectasis cases. This multiposition bed has been especially advantageous for patients too sick to use the other methods of postural drainage.

FIG. 2. Jackknife or horizontal bar drain. This easily constructed apparatus affords rapid and very satisfactory bronchial drainage and has been preferred by most ambulant patients to that shown in figure 1.

Not infrequently, however, cases are seen that require bronchoscopic aspiration as a therapeutic procedure. These cases fall into three main groups as follows:

1. Cases in which the purulent secretion cannot be completely drained posturally, due either to the thick nature of the secretion or to the weakness of the patient.
2. Cases with persistent, uncontrolled hemorrhage.

3. Cases which develop a pneumonitis about their bronchiectatic cavities, resulting usually from causes mentioned in 1. (See figures 3, 4, and 5.)

Included in the 200 cases of bronchiectasis which the writer has reviewed were 45 cases, 25 per cent, which required therapeutic bronchoscopic aspira-

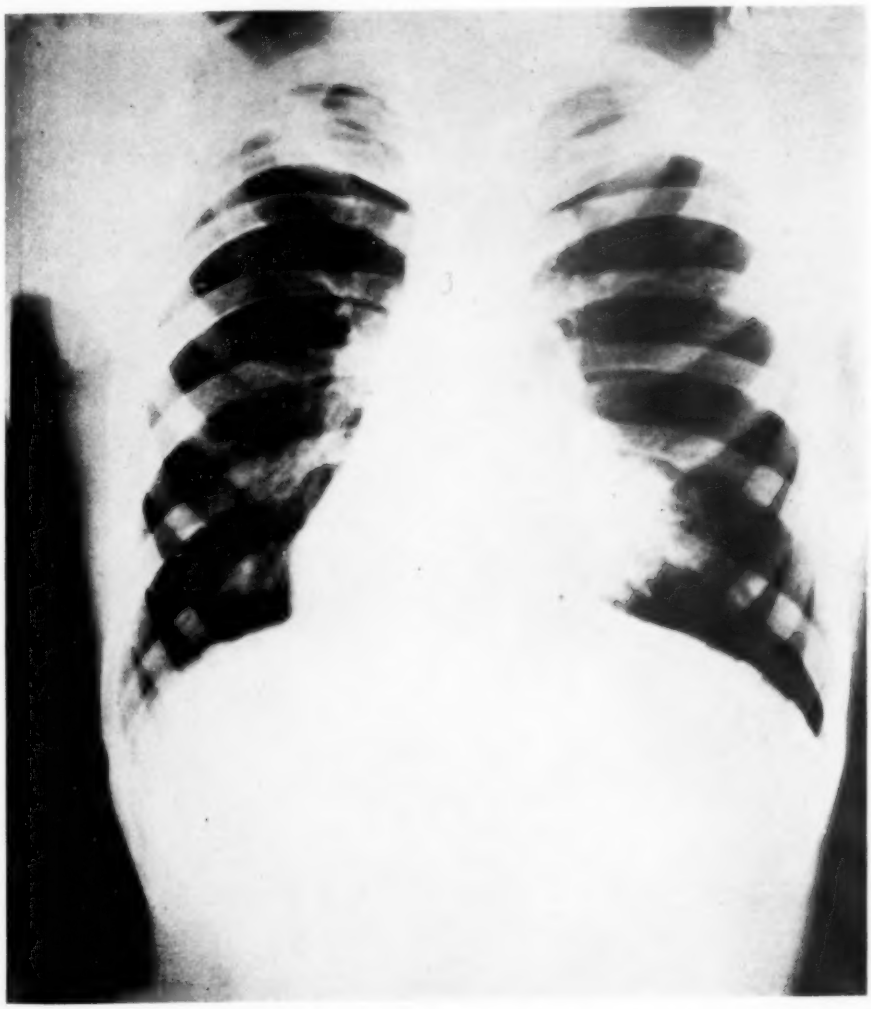

FIG. 3. Localized pneumonitis in right base, surrounding bronchiectatic dilatations. Patient discharged as "clinically cured" after several bronchoscopic aspirations combined with daily postural drainage.

tions. Of these 45 cases there were 24 which would definitely fall into group one, four cases into group two and 13 cases into group three. Many other patients were treated bronchoscopically with a view to hastening improvement and shortening the period of hospitalization, although we could not consider bronchoscopy as absolutely essential as a therapeutic measure.

We feel that bronchoscopy in the early milder cases may be beneficial as an aid in restoring the mucous membrane and its cilia to normality, thus preventing a progression of the disease and sometimes resulting in a clinical cure.

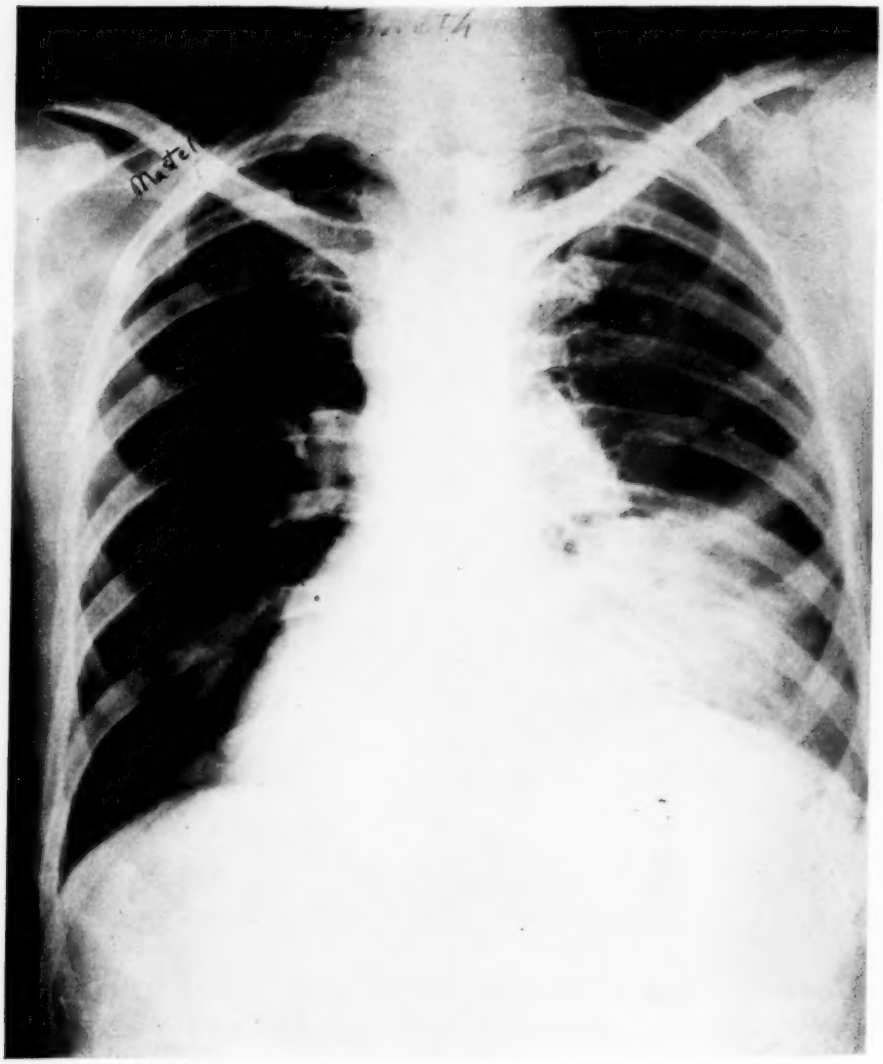

FIG. 4. Large area of pneumonitis surrounding bronchiectasis, right base (3/19/32); history briefed in article.

Bronchoscopy is important and very beneficial to group one and group two cases and often results in materially changing the aspect of a case from one of retrogression to one of clinical improvement. Cases admitted as strict bed patients are often able to be up and about in a short time and

soon feel much better, with an improvement in appetite and a gain in weight. A few bronchoscopic aspirations usually suffice in group one cases, following which they are usually able to drain sufficiently well by our ordinary methods of postural drainage. Group two cases usually have repeated small

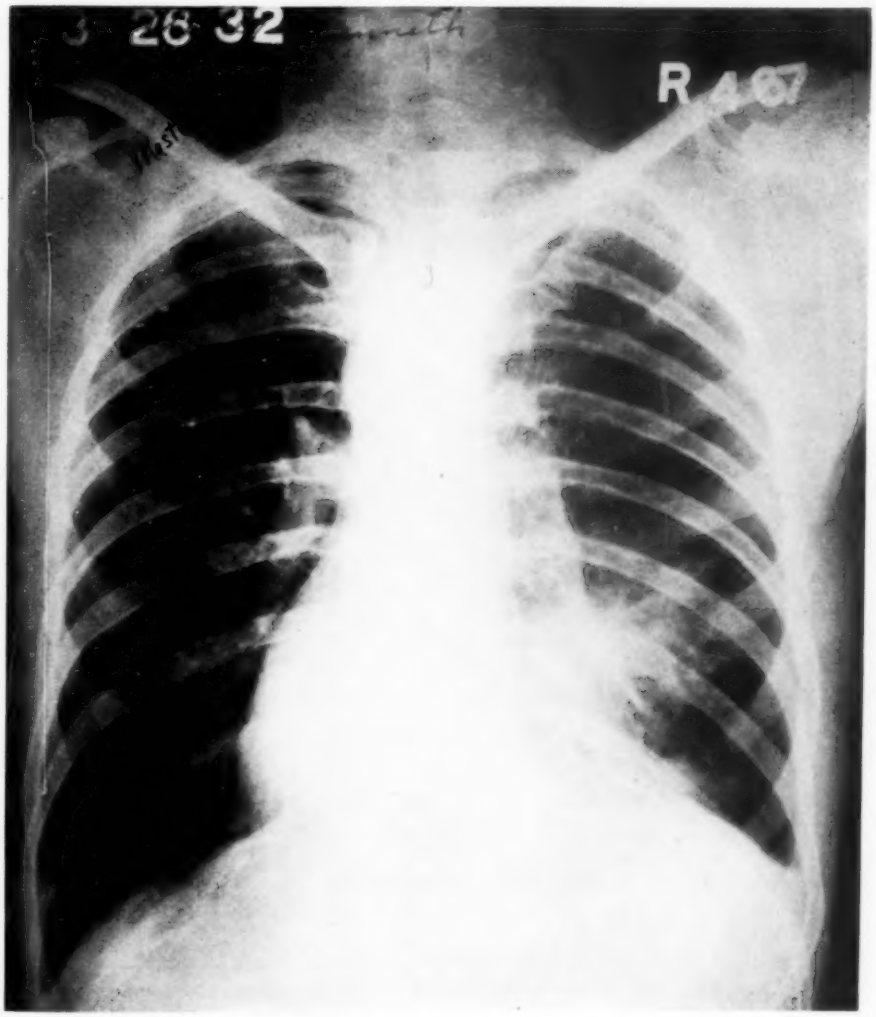

FIG. 5. X-ray of the same patient seven days later, showing the improvement following four bronchoscopic aspirations.

hemorrhages, though large hemorrhages have been reported. The bronchoscopist can usually find the bleeding area, and cauterization with silver nitrate suffices to control the bleeding.

It is the type of case seen in group three that requires early and repeated bronchoscopic treatment. These cases in which a pneumonitis has devel-

oped about the bronchial dilatations often run a toxic course with a rather high temperature and are usually progressive in severity. The inflammatory reaction or pneumonitis is usually caused by retained secretions in a severe case of bronchiectasis; or it may develop about bronchiectatic abscesses. This spread of the infection and inflammatory reaction into the pulmonary tissue is a very serious complication and requires early active treatment with a view to preventing a further spread of the pneumonitis or possible pulmonary abscess formation. We have seen several cases in which the pneumonitis continued to spread until bronchoscopic drainage was instituted, and daily bronchoscopies have been necessary in an occasional case. Semi-weekly drainage is usually sufficient in these cases to stop the spread of the infection and to start resolution of the pneumonitis and clinical improvement. One of our severe cases developed a pneumonitis followed by a pulmonary abscess and died in spite of nearly all types of treatment, including repeated bronchoscopy. Figure 3 shows an early case of unilateral bronchiectasis with pneumonitis which cleared up after repeated bronchoscopic therapy, resulting in a clinical cure.

The following case history will illustrate a not unusual sequence of events in this serious disease and how important bronchoscopic therapy is in such cases. Similar cases have been not infrequently observed in this hospital in the past three years.

Case History

Male, age 58, Retired Army Officer. Past history unimportant. Originally admitted to this hospital in 1920 for active pulmonary tuberculosis of the right upper lobe, he was readmitted for the same condition in 1921, and then later discharged as inactive as regards tuberculosis. In 1927 a diagnosis of "bronchiectasis, chronic, mild, both lower lobes," was added at this hospital. On March 11, 1932 he was readmitted to this hospital with temperature of 101° F., considerable cough and increased expectoration. He was drained posturally for nine days but his condition gradually became worse, the temperature mounted to 104° F., and the patient showed evidence of severe toxemia. A roentgenogram taken on March 19, 1932 showed considerable pneumonitis present about the large bronchiectatic dilatations (figure 4). He was given a therapeutic bronchoscopy on the morning of 3/20/32, and the temperature dropped to 99.4° F. at 4:00 p.m.; the general condition seemed much improved. About 60 c.c. of thick foul pus were aspirated from the right base at this bronchoscopy. The patient received daily therapeutic bronchoscopies on March 20, 21, 22 and 23. On the latter three days the temperature was normal in the mornings with a maximum rise to 99.6° F. in the evenings. Lessened amounts of pus were aspirated on each successive day and the patient's improvement was rapid and progressive. Further bronchoscopies were made on March 26, April 1 and 8. The patient felt very well and was temperature-free from March 24 until his discharge from hospital on April 10, 1932. Figure 5 is the roentgenogram of the chest on 3/26/32 and shows that considerable improvement had taken place since the roentgenogram on 3/19/32. Figure 6 shows that the pneumonitis had cleared on 4/8/32, two days prior to discharge from the hospital.

This patient was again admitted to the hospital on January 22, 1933, in about the same condition as on the March 1932 admission. He was given a therapeutic bronchoscopy on the day of admission. The report of the bronchoscopist was as follows:

"About 45 c.c. of pus were obtained from two cavities in the right lower lobe. The septum between the two cavities was markedly thickened and congested. Aspirated and silver nitrate applied." On January 23: "About 20 c.c. of pus found in right lower bronchus, septum much less thickened." Improvement was sudden and rapid, the patient being able to return home in a short time. He now returns to the hospital for weekly bronchoscopic therapy in addition to practicing daily postural drainage at home.

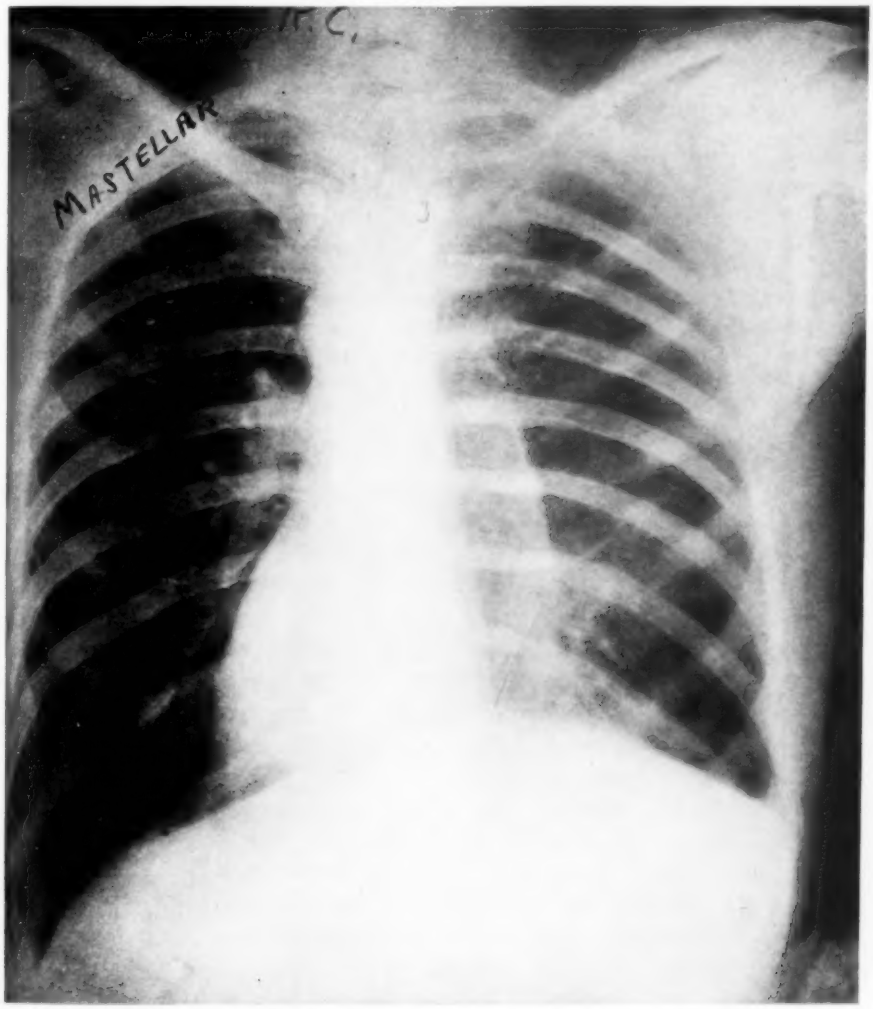

FIG. 6. Marked clearing of the pneumonitis, same patient, 4/8/32, following seven therapeutic bronchoscopic aspirations plus postural drainage several times daily.

CONCLUSIONS

Bronchoscopy is considered an important aid in the diagnosis of bronchiectasis. It is much more important and necessary from a therapeutic stand-

point, being the means by which hemorrhage may be controlled or purulent secretion aspirated in patients who are unable to drain sufficiently by our postural methods. Bronchoscopy's greatest efficacy in this disease is found in the treatment of those cases with a secondary pneumonitis about the areas of bronchiectasis. In these cases bronchoscopic aspiration may be the only effective method to prevent the spread of the inflammatory reaction with possible abscess formation and death.

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EDITORIAL

CORONARY SCLEROSIS

THE calamities associated with coronary sclerosis occupy a more dramatic and tragic place in the public consciousness today than the fatalities due to any other disease of mankind. It is not the fact that heart disease leads all other causes of death, but the sudden and often unanticipated demise of an individual with coronary sclerosis, which emphasizes the uncertain tenure of life. Scarcely a day passes but the papers chronicle the sudden death of one individual or of more than one who is preëminent in the social, political, or financial life of the community. The literature on the subject is so voluminous that a brief statement of its essentials seems justifiable.

It would appear that coronary sclerosis is on the increase, a fact as yet unproved and perhaps impossible to verify. Numerous judicial and experienced medical observers are convinced that angina pectoris and acute coronary occlusion are on the increase, and they insist that this increase is not merely an apparent one, due to increased ability of the medical profession to recognize these conditions. It is conceivable and even probable that angina pectoris, exclusive of acute coronary occlusion, has increased without any change in the incidence or degree of coronary sclerosis as a result of the increasing stress under which people live. It is noteworthy that the anginal syndrome often appears in people at a time when they have been subjected to a considerable period of mental stress.

Coronary sclerosis manifests itself in a variety of ways, the commonest of which is the syndrome of angina pectoris. In the absence of pain, it may indicate its presence by the occurrence of paroxysmal nocturnal dyspnea. The clinical picture of progressive myocardial failure, in the absence of angina pectoris or hypertension, may have its basis in coronary sclerosis. The syndrome of acute coronary occlusion may occur without any antecedent evidence that the heart was diseased. Some patients who have suffered such an accident may be remarkably free of cardiac symptoms after recovery from the acute attack. And finally, there is a group of patients who in life have never had signs or symptoms of coronary sclerosis, but who are found to have advanced degrees of coronary disease at death. Willius has designated this unrecognizable condition as occult coronary sclerosis.

The diagnosis of angina pectoris continues to offer difficulty to the medical profession. Mistakes are of two kinds: first, failure to recognize the syndrome; and second, interpretation of thoracic pain as angina pectoris when it is not. There are three characteristics of the anginal syndrome which constitute a diagnostic triad of the condition. 1. The distress is situated behind the sternum. The pain in angina pectoris may be felt else-

where in the thorax, in the neck, the jaws or the epigastrium, but if such pain is to be denoted angina pectoris, then the relationship of the symptoms must conform in all other respects to that characteristic of the disease. The term precordial pain is highly objectionable inasmuch as pain so described usually is in the apical region or in the left anterior axillary line and such pain rarely proves to be the result of coronary sclerosis. If distress on effort has its situation beneath the sternum, the presumption is that coronary disease is its cause; if the pain is in the left lateral thoracic wall, the burden is on the examiner to prove that it is angina pectoris. 2. A characteristic feature of angina pectoris is that the attack is precipitated by anything that increases cardiac work. These are, in order of importance, physical exertion, particularly walking, excitement, ingestion of food, or combinations of these factors. This essential relationship of cardiac overload to the onset of an attack is diagnostic only if the seizure stops in a few moments after the provocative factor ceases to operate. 3. The attack of angina pectoris is brief. The typical attack is over in a few minutes, seldom lasting longer than fifteen minutes. Attacks of substernal pain lasting an hour or more should always lead to the suspicion that coronary occlusion has occurred.

Angina pectoris is a diagnosis based on symptoms and not on physical signs. In from 20 to 30 per cent of patients the diagnosis must be made in spite of a negative physical examination, in the absence of any abnormality of the electrocardiogram, and without any help from roentgenographic examination of the heart. This fact assumes tremendous medicolegal importance in relation to individuals who are fraudulently seeking insurance or payments for disability. If the patient seeking insurance is dishonest enough to conceal symptoms of angina pectoris, no method of examination may disclose the existence of coronary sclerosis. If an individual seeking to establish a claim of disability feigns the classical symptoms of the disease, no medical examination may be able to prove the fraudulence of the claim.

The greatest confusion exists regarding the significance of the electrocardiographic findings in the diagnosis of angina pectoris and coronary sclerosis. Fully a third of the patients with angina pectoris have electrocardiograms that are normal in all respects. Except for the typical electrocardiographic pattern that follows acute coronary occlusion, there are no other electrocardiographic changes that positively denote coronary disease, nor will lack of changes exclude its presence. Tracings taken of patients in attacks of angina pectoris may contain transitory changes in the RS-T segment of the electrocardiogram that are almost positive evidence of insufficiency of the coronary circulation. Delays in conduction, either in the bundle of His or in the bundle branches, or a prolonged Q-wave in Lead III, as described by Pardee, are presumptive but not diagnostic evidence of coronary sclerosis. Inversion of the T-waves, except those that represent a relic of acute coronary occlusion, are not the result of chronic insufficiency

of the coronary circulation but are the result of associated cardiac pathologic changes, the commonest of which is hypertensive heart disease.

The results of treatment of coronary disease, although leaving much to be desired, compare favorably with the results of treatment of other degenerative diseases. The judicious use of nitroglycerin and amyl nitrite occupies an important place. The use of drugs of the xanthine group has afforded unmistakable relief to a number of patients. Attempts at surgical treatment of angina pectoris have been hampered by lack of knowledge of the pathways of pain from the heart, by lack of any evidence that nerve section or nerve block modified the underlying pathologic condition or the course of the disease, and by the risk of surgical procedures of the more formidable types. Fortunately the pathways of pain from the heart are becoming better understood, and surgical procedures for the relief of pain may be expected to increase in precision and effectiveness proportionately. Needless to say, such operations must be reserved for patients whose pain has remained intractable following adequate medical treatment. Total removal of the thyroid gland has been reported to afford relief to patients with angina pectoris, and this method of surgical treatment calls for further careful evaluation.

Finally, there is the most important consideration of rest. The prescription of rest should be most accurate and individualized in every case. It should be planned to produce adequate reduction of metabolic demands and of physical and mental stress.

We, as physicians, are implored to do something to reduce the ravages of coronary sclerosis. But how much can we do? After all, coronary sclerosis is a part of senescence, and senescence is as much a part of life as birth and growth, and just as inevitable. Pneumonia has been called the friend of the aged, and death from coronary disease has just as much right to that designation. Moreover, sudden death spares the patient the suffering and invalidism often associated with death from other causes. On the other hand, everyone would like to find a way to prevent death from coronary sclerosis in the fifth and sixth decades; but the tempo and demands of life for the ambitious do not lend themselves to easy adjustment. Now, more than ever before, life for the average individual is a contest and a battle of energy. This is as true of nations as it is of individuals. The person who decides to indulge in the luxurious attitude of "dolce far niente" is apt to find himself outdistanced. To ask people to give up their stress in life is like crying, "Peace, peace, when there is no peace." When the social structure and ideals of our civilization will allow individuals to live leisurely and to survive, then we may anticipate some amelioration of coronory sclerosis. Until then we must content ourselves with such makeshifts of relaxation and leisurely living as our struggle for existence will permit.

A. R. Barnes.

REVIEWS

Clio Medica: A Series of Primers on the History of Medicine. Edited by E. B. KRUMBHAAR, M.D. *Volume X. Nutrition.* By GRAHAM LUSK, Sc.D., M.D., LL.D. xi + 142 pages; 11.5 x 17 cm. Paul B. Hoeber, New York. 1933. Price, $1.50.

This is a little book no student of medicine, young or old, can afford to miss. It traces through the centuries the development of our knowledge of nutritional metabolism—with scholarly care, and with a balanced judgment of relative values which could be given only by a master in this field. The reader, however, is not kept in the pure ether of mental processes. The picturesque and personal sides of the great scientists' lives are woven into the tale; and there is still space for quotation, for honest admiration, for anecdotes by the way, for keen comments, for comparisons with the science of our day and its patrons. A lesser man might have written a valuable tome on this subject but it took a great man to write this little book.

M. C. P.

Diabetes Mellitus, A Handbook of Simplified Methods of Diagnosis and Treatment. By I. M. RABINOWITCH, D.Sc., M.D., C.M., F.R.C.P.(C.). xv + 246 pages; 15 x 21 cm. The Macmillan Co., of Toronto, Canada. 1933. Price, $3.50.

The author's wide experience in the Clinic for Diabetes of the Montreal General Hospital has led him to feel that an effective treatment for diabetes must be simple, both for the patient to follow and for the physician to control. One of the chief purposes of his book is to portray a method of treatment, in which the patient need know very little of food values and can substitute household measures and portion models for scales, and which the physician can control without determinations of blood sugar or alkali reserve. Only by such simplifications does the author believe that a good average of results can be obtained in the treatment of diabetics in general practice. Conceding that ideal treatment would involve more exact determinations, he does not feel that ideal treatment is capable of wide application outside of hospitals.

The methods used for instructing the patient in the measurement of the diet are ingenious and practical. The descriptions of the tests for sugar and of the procedures for the differentiation of glucose from other urinary reducing substances are clear and detailed. It seems to the reviewer, however, that in advocating the fermentation test as a quantitative procedure, instead of a titration method, the author is not simplifying but complicating the physician's task. It seems unwise also to advocate so much reliance upon urinary sugar and not to stress more than is done the fact that in a very large number of cases a heightened renal threshold permits marked hyperglycemia to exist without glycosuria.

The author employs a high carbohydrate-low calorie diet. An average adult diet would contain approximately 275 grams carbohydrate, 45 grams fat, and 80 grams protein, yielding about 1,800 calories. A portion of the carbohydrate allowance is often given in sweets such as jams and marmalade. On the other hand, fats are severely restricted. The patient's weight is kept at about five pounds below the normal for the age, sex, and height as determined by a formula. Insulin is employed when, in spite of this maintenance diet, sugar persists in the urine or hyperglycemia exists. The results of this system of treatment in the author's clinic have proved very satisfactory. These results are contained in other publications but are not included in this book.

There are brief chapters on the various clinical types of diabetes, its complica-

tions, and its relation to arteriosclerosis, gangrene, tuberculosis, thyroid disease, eye lesions, skin lesions, etc.

The book is not written primarily for internists but for general practitioners as well. It has been developed from the author's lectures to fourth year students. It is short, clear, definite, and practical. If in some respects the author's opinions seem radical, they are the more stimulating. For physicians who wish to give a trial to the author's theory of the dietetic treatment of diabetes, his book will serve as an excellent manual. It is for this purpose that it was written. Perhaps at some later date the author will collect in another volume the data which would be of interest to those who would wish critically to analyze his results.

M. C. P.

The Colon, Rectum, and Anus. By FRED W. RANKIN, B.A., M.A., M.D., F.A.C.S.; J. ARNOLD BARGEN, B.S., M.D., M.S. in Medicine, F.A.C.P.; and LOUIS A. BUIE, B.A., M.D., F.A.C.S. 812 pages; 435 illustrations. W. B. Saunders Company, Philadelphia. 1932.

This monograph is based on the large experience of The Mayo Clinic. Though written largely from the surgical point of view, it contains a great deal of interest and of value to the internist. This is especially true of certain chapters, notably those on granulomatous diseases, chronic ulcerative colitis, parasitic diseases, colonic manifestations of systemic origin, and functional obstructive disorders. Of these, the chapter on ulcerative colitis is particularly instructive and presents in a very thorough manner not only the clinical and pathological aspects of the disease and its complications but also an admirable summary of the investigations which have led Bargen to attribute etiological importance to a diplococcus. The description of his technic will be of interest to those attempting to confirm his work. The other medical chapters contain less evidence of originality. On the other hand, the chapters on the neoplasms, benign and malignant, of the colon and rectum are important contributions to the literature of this subject. Careful studies are presented of the clinical symptoms and signs, of roentgenological methods of investigation and of mortality. The various operative procedures are carefully evaluated.

The book is for the most part clearly written and very well illustrated. It will be an important reference volume for the internist as well as the surgeon.

M. C. P.

The Psychological Effects of Oxygen Deprivation (Anoxemia) on Human Behavior. By Ross C. MCFARLAND, Ph.D., Columbia University. 127 pages. Columbia University Press, New York. 1932. Price, $1.50.

This monograph thoroughly discusses a problem which not infrequently comes to the attention of general physicians, especially in this age of aviation when persons suffer from the lack of oxygen as the result of high altitudes.

McFarland completely reviews the literature, gives a bibliography of 184 items, and discusses the experimental work carried out. His experimentations conclusively show the effects of oxygen upon human behavior. "Climate seems to have an important influence in determining the character and productiveness of a people as well as their physical characteristics. . . . The mental and emotional differences between the races of climatic extremes are as striking as the physiological and cultural productiveness."

Simple sensory and motor responses are not seriously impaired until extreme anoxemia is present. Volitional reactions are more easily impaired under acute anoxemia and loss of motor control is then marked although there are great individual differences. Loss of memory occurs very early, and the effect of diminished oxygen upon attention is very definite. The higher mental processes are impaired

and the basic patterns of personality can be obtained. Emotional extremes are more easily induced in anoxemia. "The results indicate clearly that personality is in the final analysis dependent upon the physio-chemical processes and that more thorough or profound knowledge concerning human behavior may be obtained by combining psychological and biochemical research."

J. L. McC.

Bacterial Infection with Special Reference to Dental Practice. By J. L. T. Appleton, Jr., B.S., D.D.S., Professor of Microbiology and Bacteriopathology, The Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania. Second edition. 654 pages with 122 engravings and 4 colored plates. Lea and Febiger, Philadelphia. 1933.

The general plan of this edition is very similar to that of the first. The book is divided into three parts. The first deals briefly with the morphology, physiology, and classification of bacteria, with their relation to their environment and with the action of chemicals upon them. The second part discusses rather thoroughly the subjects of infection and immunity. The third is devoted to various infections of the oral cavity. The book, however, has been in large part rewritten and much new material has been added. The second part has been much enlarged and the order in which the various topics are discussed has been rearranged. The treatment of oral hygiene has been extended and an entire chapter has been given to its discussion. The third part shows considerable revision and several new chapters have been added. The chapter originally given over to the discussion of clinical bacteriology has been divided and the several parts each appended in its appropriate place. A very useful list of references is given. The purposes of this book, as stated by the author, are: (1) "To aid the reader to form a comprehensive concept of infection"; and (2) "To point out wherever a knowledge of infection will help the dentist in understanding or solving his problems." These purposes would seem to be adequately met. The book is primarily of value to the dental student and the dentist. However, much of the third section should also be of interest to the physician.

F. W. H.

COLLEGE NEWS NOTES

Acknowledgment is made of the following gifts of publications to the Library by members of the College:

Dr. I. D. Bronfin (Fellow), Denver, Colo.—one reprint;
Dr. Philip B. Matz (Fellow), Washington, D. C.—one reprint;
Dr. Adolph Sachs (Fellow), Omaha, Nebr.—one reprint;
Dr. John W. Shuman (Fellow), Los Angeles, Calif.—one reprint;
Dr. Martin J. Synnott (Fellow), Montclair, N. J.—one reprint;
Dr. William H. Walsh (Fellow), Chicago, Ill.—one reprint;
Dr. Hyman I. Goldstein (Associate), Camden, N. J.—one reprint.

At the Annual Meeting of the Medical Society of Virginia, during October, Dr. F. H. Smith (Fellow), Abingdon, Va., was made President-Elect, and Dr. James K. Hall (Associate), Richmond, Va., was elected a Vice-President.

Dr. John I. Marker (Fellow), Davenport, Iowa, was elected President of the Southeastern District Medical Society of Iowa during October, 1933.

Dr. Edward M. Green (Fellow) has resigned as Superintendent of the Harrisburg State Hospital, Harrisburg, Pa., after sixteen years of service in that capacity.

The City of Philadelphia, Trustee under the will of John Scott of Edinburgh, through its Board of Directors of City Trusts, has awarded The John Scott Medal to George Richards Minot in recognition of his work for the development of the liver treatment of pernicious anemia.

On October 23, 1933, Dr. Minot gave the first Jessie Horton Koessler Lecture of the Institute of Medicine of Chicago, his subject being "Anemia: Etiology and Treatment"; he also gave a lecture on this subject before the Cleveland Academy of Medicine on November 17, 1933.

Dr. Walter Freeman (Fellow), Washington, D. C., resigned December 1, 1933, as Director of Laboratories at St. Elizabeth's Hospital, and will devote himself to university and clinic work and to the private practice of neurology. Dr. Freeman addressed the Montgomery Co. (Ohio) Medical Society at Dayton recently on "Psychological Plagues." He was recently reëlected Secretary of the Section on Nervous and Mental Diseases of the Medical Society of the District of Columbia. His present address is 1726 Eye Street, Washington, D. C.

Dr. George A. Pemberton Wright (Fellow), formerly of Kingston, Jamaica, is now located in Delancey, Guernsey, Channel Islands.

Dr. Albert E. Russell (Fellow), formerly of the United States Bureau of Mines, Washington, D. C., is now in charge of the public health and medical program of the

Tennessee Valley Authority, Knoxville, Tenn. Programs are being organized for malaria control, pellagra prevention, trachoma clinics, venereal disease clinics, tuberculosis, maternal and child welfare.

Dr. J. C. Meakins (Fellow), Montreal, and Dr. Cyrus C. Sturgis (Fellow), Ann Arbor, Mich., will be guest speakers at the Dallas Southern Clinical Society meeting, March 26 to 30, 1934.

Dr. Adolph Sachs (Fellow), Omaha, Nebr., was the first President of the newly organized Omaha Mid-West Clinical Society—"to bring post-graduate opportunities to the back door of the Physicians for the least cost." The first Annual Assembly of this organization was held October 30 to November 3, 1933, in Omaha. Among guest speakers were the following Fellows of the College: Dr. James B. Herrick and Dr. Julius H. Hess, both of Chicago.

ERRATUM.—In the ANNALS OF INTERNAL MEDICINE for December 1933, page 739, line 27, *mercurial* should read *compound* or *molecule*.

OBITUARIES

DR. JOHN C. S. LAPPEUS

On November 17, 1933, occurred the death of Dr. John C. S. Lappeus, of Binghamton, N. Y., following a short period of illness.

Dr. Lappeus was born in 1878 in Hornellsville, N. Y., now known as Hornell. He received his preliminary education at the Keystone Academy, Factoryville, Pa., and was granted his degree of M.D. in 1904 by the University of Buffalo School of Medicine. He was licensed to practice in both New York and Pennsylvania. For many years he was Attending Physician and member of the staff of the City Hospital, Lourdes Hospital and the Susquehanna Valley Home, all of Binghamton. He was a member of the staff on health education of the Binghamton Public Schools, a member of the Binghamton Academy of Medicine, the Broome County Medical Society, the New York State Medical Society and the American Medical Association. On December 30, 1926, he was elected to Fellowship in the American College of Physicians.

The American College of Physicians, in common with other professional and lay organizations, to many of which Dr. Lappeus belonged, has lost in his death a very able, loyal, earnest and progressive worker.

H. D. Marvin, M.D., F.A.C.P.,
Binghamton, N. Y.

DR. FRANK PERKINS KEYES

Dr. Frank Perkins Keyes (Associate), Brooklyn, N. Y., died suddenly August 18, 1933, at Sudbury, Vt., of cerebral hemorrhage and diabetes mellitus.

Dr. Keyes was born February 3, 1857. He graduated from the Long Island College Hospital in 1888 and practiced medicine, therefore, for more than fifty years. After graduation, he became assistant to the late Dr. Stephen Edward Fuller, who was a surgeon. He continued this work until Dr. Fuller's death, and then continued general practice, doing some surgical work. He belonged to the type of "old fashioned doctors"; he brought common sense and general ability to his problems. He was quiet and unassuming but held in the highest esteem by his patients. His unusual kindness and sympathy were the outstanding features of his character and practice.

Dr. Keyes was a member of the Medical Society of the County of Kings, the New York State Medical Society, a Fellow of the American Medical Association, and had been an Associate of the American College of Physicians from its beginning.

DR. NOBLE PRICE BARNES

Dr. Noble Price Barnes (Fellow), Washington, D. C., died November 26, 1933; aged 62 years.

Dr. Barnes graduated from the Baltimore Medical College in 1893. He occupied the Chair of Pediatrics in the National Medical College, which later became the George Washington University Medical School. He taught Materia Medica in both the Dental and Medical Departments of George Washington University.

Dr. Barnes was a former President of the Medical Association of the District of Columbia, a former Secretary, President and member of the Board of Governors of the American Therapeutic Society, an organizer and former President of the Washington Medical and Surgical Society, and was one of the first Fellows of the American College of Physicians, having been elected in 1916. He was a delegate to the United States Pharmacopeia Convention in 1930.

" His generous spirit and geniality will be missed by all with whom he came in contact."

WALLACE M. YATER, M.D., F.A.C.P.,
Governor for the District of Columbia.

ABSTRACT OF MINUTES OF THE BOARD OF REGENTS

PHILADELPHIA, PA.

December 3, 1933

The Board of Regents of the American College of Physicians met at the College Headquarters at Philadelphia at 10:30 a.m., December 3, 1933; presided over by the President, Dr. George Morris Piersol.

The following members of the Board of Regents were present: Dr. George Morris Piersol, Dr. Charles G. Jennings, Dr. James H. Means, Dr. Jonathan C. Meakins, Dr. William D. Stroud, Dr. William Gerry Morgan, Dr. James Alex. Miller, Dr. Sydney R. Miller, Dr. David P. Barr, Dr. James B. Herrick, Dr. Clement R. Jones, Dr. S. Marx White, Dr. Walter L. Bierring, Dr. John H. Musser, Dr. O. H. Perry Pepper, Dr. Francis M. Pottenger, Dr. Luther F. Warren, Dr. Maurice C. Pincoffs and Dr. Ernest B. Bradley. Dr. Charles H. Cocke, a member of the Committee on Credentials, was present as a guest, and the Executive Secretary, Mr. E. R. Loveland, acted as Secretary of the meeting.

The Executive Secretary read abstracted Minutes of the preceding meeting of the Board of Regents at Montreal, February 10, 1933, which were approved as read.

President Piersol commented briefly concerning the fact that the work of the College had been going along in a suitable manner and that no previous meeting of the Board of Regents, or of the Executive Committee, had seemed necessary, especially in view of the desirability of economy in the administration of the College.

REPORT OF THE SECRETARY-GENERAL

Dr. William Gerry Morgan, Secretary-General, presented the following communications:

A letter from the Chairman of the Committee on Cost of Current Medical Periodicals, thanking the College for its endorsement and assistance in their recent efforts to reduce and stabilize subscription prices of German periodicals particularly;

Two communications from Fellows applying for reinstatement. By resolution, Dr. Charles Jack Hunt, New York, N. Y., and Dr. Eugene A. Gilbert, Chattanooga, Tenn., were reinstated as Fellows of the College;

A number of letters from Fellows and Associates concerning resignations. By individual resolution, the following resignations were accepted:

Fellows:

I. Hope Alexander, Pittsburgh, Pa.
John B. Hawes, 2nd, Boston, Mass.

Associates:

Roderick E. Albright, Allentown, Pa.
James W. Bruce, Louisville, Ky.
David B. Davis, Grand Rapids, Mich.
Charles A. Howland, Schenectady, N. Y.
Glendon R. Lewis, Syracuse, N. Y.
John C. E. Nielsen, Los Angeles, Calif.
E. A. Sutherland, Madison, Tenn.

A number of letters from Fellows and Associates concerning fees and dues, which were acted upon individually according to merits of the cases and provisions of the By-Laws.

Dr. William Gerry Morgan, Secretary-General, then read the following list of deaths reported to the College since the last meeting of the Board of Regents:

Fellows:

S. Franklin Adams, New York, N. Y.	September 28, 1933
Neil Andrews, Oshkosh, Wis.	March 10, 1933
Noble P. Barnes, Washington, D. C.	November 26, 1933
William R. Bathurst, Little Rock, Ark.	August 30, 1933
John S. Davis, University, Va.	September 21, 1933
George J. Eckel, Buffalo, N. Y.	October 29, 1933
Thomas Wray Grayson, Pittsburgh, Pa.	May 16, 1933
Neal L. Hoskins, Detroit, Mich.	July 21, 1932
John C. S. Lappeus, Binghamton, N. Y.	November 17, 1933
Martin J. Larkin, Toledo, Ohio	March 11, 1933
R. E. Loucks, Detroit, Mich.	June 5, 1933
Henry G. Mehrtens, San Francisco, Calif.	February 28, 1933
Edward W. Meis, Sioux City, Iowa.	December 7, 1932
William H. Mercur, Pittsburgh, Pa.	July 16, 1933
Charles Howard Miller, San Leandro, Calif.	February 2, 1933
Franklin E. Murphy, Kansas City, Mo.	February 20, 1933
Edward O. Otis, Exeter, N. H.	May 28, 1933
Judd Campbell Shellito, Independence, Iowa	April 16, 1933
W. Blair Stewart, Atlantic City, N. J.	July 11, 1933
Shannon Laurie Van Valzah, Denver, Colo.	July 11, 1933
John Bloss Wolfe, Wilkes-Barre, Pa.	June 1, 1933

Associates:

Raymond J. Harris, Philadelphia, Pa.	August 9, 1933
Frank Perkins Keyes, Brooklyn, N. Y.	August 18, 1933
Thomas J. McKinney, Champaign, Ill.	May 27, 1933
W. F. Mitchell, Superior, Nebr.	June 3, 1932
Walker Eugene Stallings, Denver, Colo.	May 6, 1933

By resolution, the following were discontinued from the roll of the College:

Ray C. Barrick (Associate), Joliet, Ill.
George Henry Benton (Fellow), Coral Gables, Fla.
Julian R. Blackman (Fellow), Salt Lake City, Utah
James E. Harvey (Associate), Pasadena, Calif.
Finn Koren (Associate), Watertown, S. D.

Report of the General Chairman of the Clinical Session

Dr. James B. Herrick, General Chairman of the Eighteenth Annual Clinical Session, reported upon preliminary arrangements made by him as General Chairman of the Session. He has appointed Dr. Arthur R. Elliott as Chairman of the Committee on Clinics, and has selected certain other committee members. In conference with Dr. Elliott, it has been decided that only the medical schools and the teaching hospitals will be called upon to coöperate in presenting clinics. A member of the Committee on Clinics has been selected from each of these medical schools and hospitals, including the Graduate School of Medicine of the University of Chicago, the University of Illinois College of Medicine, Northwestern University Medical School, Loyola University School of Medicine, Rush Medical College, Presbyterian Hospital, Michael Reese Hospital, St. Luke's Hospital, Mercy Hospital, Children's Memorial Hospital

and the Cook County Hospital. Dr. Herrick particularly asked for suggestions from the Board of Regents concerning the number of guest clinicians that should be invited.

In connection with an entertainment program for the visiting women, Dr. Herrick said that the Ladies Committee would provide headquarters at the hotel for the registration and reception of the ladies. A directory of interesting places and events will be furnished, and possibly one or two organized trips will be arranged.

Dr. Herrick discussed the names of several men of national reputation as the chief speaker on the occasion of the annual banquet. In connection with publicity for the meeting, Dr. Herrick reported that an article on "Chicago as a Medical Center" had been prepared and submitted to the Editor of the ANNALS OF INTERNAL MEDICINE for publication. There was general discussion of the subject of publicity concerning the program. It was the consensus of opinion of the Board of Regents that increased effort should be made toward improving the character and scope of the publicity concerning the scientific program. The Executive Secretary was called upon to explain the present system of promoting publicity, which briefly has been as follows: a local committee of approximately five members, all physicians and Fellows of the College, has been selected to coöperate with various news agencies and local reporters. Preceding the meeting, abstracts of as many papers as possible are secured, and these are further translated for the use of the reporters. During the meeting at least one of these physicians is in attendance at each session, and thereafter translates the papers in understandable language to the reporters, thus giving the reporters invaluable aid and assuring to the College a better type of publicity than could be secured from lay reporters. It has been customary to have in attendance representatives from Science Service, the Associated Press, the United Press, and the local newspapers. A regular headquarters office with typewriters and a secretary has been provided.

There was general discussion concerning the matter of guest clinicians, some expressing the opinion that most of the members would be more particularly interested in seeing what is being done by the men in Chicago, although the addition of a few guest clinicians from outside of Chicago would undoubtedly be very appropriate and would attract additional attendants to the clinics. It was further stated that the clinical program should be as broad as possible, and that the work in some of the great laboratories in Chicago should particularly be shown.

Dr. Herrick discussed the arrangements for the Smoker, the first evening of the Session, expressing the desire to have an entertaining though dignified program.

Dr. George Morris Piersol, President, distributed the preliminary copy of the program for the General Sessions, for which he is responsible. He had selected, for the most part, Fellows of the College, expressing the opinion that there are an adequate number of eminent authorities within the College membership without inviting a large number of guests. The speakers have been selected from all parts of the country, and the program will include a broad scope of subjects. Special papers have been arranged for those interested in pediatrics, neurology, psychiatry and various other specialties affiliated with internal medicine. Radiology will be taken care of largely through special clinics this year.

Dr. James H. Means expressed the opinion that the addition of one or two distinguished foreign guests always adds much to the meeting.

Mr. E. R. Loveland, Executive Secretary, reported that the usual business arrangements had been made for the Clinical Session, that the Palmer House had been selected for headquarters, where there would be adequate facilities for the general headquarters, for the exhibits, for the General Sessions and for accommodation of practically all physicians who may desire to stay at the headquarters hotel.

Report of the Committee on Credentials

Dr. Sydney R. Miller, Chairman of the Committee on Credentials, reported that his Committee had examined carefully the credentials of all candidates for Fellowship and Associateship. Of 55 candidates for Fellowship, 39 were recommended for election to Fellowship, 1 was recommended for election first to Associateship, 6 were held as Associates, 5 were deferred for further information and 4 were recommended for rejection. Of the 110 candidates for Associateship, 92 were recommended for election to Associateship, 6 were deferred for more information and 12 were recommended for rejection.

Dr. Miller then presented the following list of 40 candidates recommended to the Board of Regents for election to Fellowship: (1, indicates proposer; 2, seconder; 3, endorser).

California

Dudley Wayne Bennett, San Francisco.
1. Ernest H. Falconer; 2. Irwin C. Schumacher; 3. William J. Kerr and Hans Lisser.

Louis Henry Fales, Livermore.
1. Audley O. Sanders; 2. M. B. Marcellus; 3. William J. Kerr and Hans Lisser.

Arthur Max Hoffman, Los Angeles.
1. Harold H. Smith; 2. Henry H. Lissner; 3. Egerton Crispin.

Earl Oriol Gregor Schmitt, San Jose.
1. D. Schuyler Pulford; 2. Albert H. Rowe; 3. Walter L. Bierring and Hans Lisser.

Milo Kenney Tedstrom, Santa Ana.
1. Roland Cummings; 2. R. Manning Clarke; 3. Egerton Crispin.

Connecticut

William Haviland Morriss, Wallingford.
1. Arthur Bliss Dayton; 2. George Blumer; 3. Henry F. Stoll.

Illinois

David Oscar Nathaniel Lindberg, Decatur.
1. Cecil M. Jack; 2. O. O. Stanley; 3. Samuel E. Munson.

Edgar McLean Stevenson, Bloomington.
1. Gerald M. Cline; 2. John R. Vonachen; 3. Samuel E. Munson.

Indiana

William G. Crawford, Terre Haute.
1. Herman M. Baker; 2. Edgar F. Kiser; 3. Robert M. Moore.

Leon G. Zerfas, Indianapolis.
1. J. A. MacDonald; 2. Edgar F. Kiser; 3. Robert M. Moore.

Kentucky

John Sharpe Chambers, Lexington.
1. Charles N. Kavanaugh; 2. W. S. Wyatt; 3. Ernest B. Bradley.

Louisiana

Robert Theodore Lucas, Shreveport.
1. Arthur A. Herold; 2. T. P. Lloyd; 3. Joseph E. Knighton.

Maine

John O. Piper, Waterville.
1. E. H. Drake; 2. E. R. Blaisdell; 3. E. W. Gehring.

Michigan

Roy DeVaughan Metz, Detroit.
1. Alpheus F. Jennings; 2. Herman H. Riecker; 3. James D. Bruce.

John William Towey, Powers.
1. J. A. Myers; 2. Stuart Pritchard; 3. James D. Bruce.

Minnesota

Robert Bernard Radl, Minneapolis.
1. J. A. Myers; 2. Arnold S. Anderson; 3. S. Marx White.

New York

Alvan LeRoy Barach, New York.
1. Joseph H. Barach; 2. Walter W. Palmer; 3. James Alex. Miller and Robert A. Cooke.

Clifton H. Berlinghof, Binghamton.
1. H. B. Marvin; 2. Walter A. Baetjer; 3. Robert A. Cooke.

George Walter Cramp, Brooklyn.
1. Frank Bethel Cross; 2. Charles Eastmond; 3. Luther F. Warren and Robert A. Cooke.

Edward Percy Eglee, New York.
1. Grant Thorburn; 2. J. Burns Amberson; 3. James Alex. Miller and Robert A. Cooke.

Eugene Roland Marzullo, Brooklyn.
1. John B. D'Albora; 2. A. F. R. Andresen; 3. Luther F. Warren and Robert A. Cooke.

Foster Murray, Brooklyn.
1. Julian P. Dworetzky; 2. Eugene S. Dalton; 3. James Alex. Miller and Robert A. Cooke.

North Carolina

Coy Cornelius Carpenter, Wake Forest.
1. L. B. McBrayer; 2. William B. Dewar; 3. C. H. Cocke.

Frederick Moir Hanes, Durham.
1. Wingate M. Johnson; 2. T. C. Redfern; 3. C. H. Cocke.

Daniel Franklin Milam, Raleigh.
1. Verne S. Caviness; 2. Hubert B. Haywood; 3. C. H. Cocke.

James William Vernon, Morganton.
1. Wingate M. Johnson; 2. Coite L. Sherrill; 3. C. H. Cocke.

Oklahoma

Ben Hunter Cooley, Norman.
1. L. J. Moorman; 2. Wann Langston; 3. Lea A. Riely.

Hugh Gilbert Jeter, Oklahoma City.
1. J. T. Martin; 2. Arthur W. White; 3. Lea A. Riely.

Pennsylvania

Samuel Goldberg, Philadelphia.
1. John A. Kolmer; 2. Edward Weiss; 3. E. J. G. Beardsley.

Thomas Murphy McMillan, Philadelphia.
1. Olin S. Allen; 2. Alfred Stengel; 3. E. J. G. Beardsley.

James MacLaren Strang, Pittsburgh.
1. Frank A. Evans; 2. R. R. Snowden; 3. E. Bosworth McCready.

Texas

Robert Mitchell Barton, Dallas.
1. H. M. Winans; 2. G. E. Brereton; 3. C. T. Stone.

Medical Corps, U. S. Army

Major Frank Henry Dixon, Corozal, Canal Zone.
1. C. D. Briscoe; 2. J. F. Siler; 3. William M. James and Robert U. Patterson.

Medical Corps, U. S. Navy

Admiral Perceval Sherer Rossiter, Washington, D. C.
1. Charles E. Riggs.

Canada

Ontario

George K. Wharton, London.
1. George E. Brown; 2. L. G. Rowntree; 3. Jabez H. Elliott.

Quebec

James Bertram Collip, Westmount.
1. A. T. Henderson; 2. C. G. Sutherland; 3. D. Sclater Lewis.

Mexico

Ignacio Chavez, Mexico City.
1. Lee Rice; 2. Joe Kopecky; 3. C. T. Stone.

Francisco de P. Miranda, Mexico City.
1. Lee Rice; 2. Joe Kopecky; 3. C. T. Stone.

Siam

Edwin Charles Cort, Chiengmai.
1. Thomas R. Brown; 2. Ernest H. Gaither; 3. Henry M. Thomas, Jr.

On motion seconded and regularly carried, it was

RESOLVED, that the above 39 candidates, individually presented, shall be and are herewith elected to Fellowship in the American College of Physicians.

Dr. Miller then presented the following list of 93 candidates recommended for election to Associateship: (1, indicates proposer; 2, seconder; 3, endorser).

Arizona

Jesse Dewey Hamer, Phoenix.
1. Charles S. Kibler; 2. Samuel H. Watson; 3. W. Warner Watkins.

Arkansas

Elmer John Munn, El Dorado.
1. Fergus O. Mahoney; 2. George B. Fletcher; 3. Oliver C. Melson.

California

William Clifford Cooke, San Diego.
1. Clair L. Stealy; 2. Lyell C. Kinney; 3. Egerton Crispin.

Felix Cunha, San Francisco.
1. William J. Kerr; 2. Fred H. Kruse; 3. Hans Lisser.

Cullen Ward Irish, Los Angeles.
1. Stephen Smith; 2. Charles W. Thompson; 3. Egerton Crispin.

Raymond Arthur Sands, Santa Monica.
1. John V. Barrow; 2. Roland Cummings; 3. Egerton Crispin.

James Robert Sanford, Pasadena.
1. Robert Eward Ramsay; 2. F. M. Pottenger; 3. Egerton Crispin.

Rudolph Herbert Sundberg, San Diego.
1. Addison E. Elliott; 2. James F. Churchill; 3. Egerton Crispin.

Connecticut

Marcus Backer, Bridgeport.
1. Daniel P. Griffin; 2. George Blumer; 3. Henry F. Stoll.

Abe S. Brown, Waterbury.
1. John H. Foster; 2. J. Harold Root; 3. Henry F. Stoll.

Henry Caplan, Meriden.
1. Thomas P. Murdock; 2. Joseph I. Linde; 3. Henry F. Stoll.

John Cowles White, New Britain.
1. G. Gardiner Russell; 2. Orin R. Witter; 3. Henry F. Stoll.

District of Columbia

John Minor, Washington.
1. Alexander B. Moore; 2. Lester Neuman; 3. Wallace M. Yater.

Florida

Kenneth Phillips, Miami.
1. William Henry Watters; 2. C. F. Roche; 3. James B. Herrick.

Georgia

John Cox Wall, Eastman.
1. Thomas E. Rogers; 2. James A. Fountain; 3. Russell H. Oppenheimer.

Illinois

Stuart Welsh Adler, Rock Island.
1. William F. Schroeder; 2. Hugh A. Beam; 3. Samuel E. Munson.

Thomas Davis Masters, Springfield.
1. Frank Parsons Norbury; 2. Frank G. Norbury; 3. Samuel E. Munson.

Maxim Pollak, Peoria.
1. William Henry Walsh; 2. George W. Parker; 3. Samuel E. Munson.

Indiana

Harry Brandman, Whiting.
1. Paul H. Dietrich; 2. J. A. Teegarden; 3. Robert M. Moore.

James H. Stygall, Indianapolis.
1. Edgar F. Kiser; 2. John A. MacDonald; 3. Robert M. Moore.

Iowa

William Edward Ash, Council Bluffs.
1. Ernest Kelley; 2. A. A. Johnson; 3. Walter L. Bierring and Tom Bentley Throckmorton.

Robert N. Larimer, Sioux City.
1. John H. Peck; 2. A. C. Page; 3. Walter L. Bierring and Tom Bentley Throckmorton.

Theodore John Pfeffer, DeWitt.
1. J. Arnold Bargen; 2. Henry W. Woltman; 3. George E. Brown, Tom Bentley Throckmorton and Walter L. Bierring.

Benjamin Franklin Wolverton, Cedar Rapids.
1. John H. Peck; 2. Addison C. Page; 3. Walter L. Bierring and Tom Bentley Throckmorton.

Kentucky

Benjamin Lane Brock, Waverley Hills.
1. Lawrason Brown; 2. Edgar Mayer; 3. Ernest B. Bradley.

Carl Hale Fortune.
1. Charles N. Kavanaugh; 2. George H. Wilson; 3. Ernest B. Bradley.

Louisiana

Thomas Everett Strain, Shreveport.
1. W. S. Kerlin; 2. T. E. Williams; 3. J. E. Knighton.

Clarence Hungerford Webb, Shreveport.
1. T. P. Lloyd; 2. C. P. Rutledge; 3. J. E. Knighton.

Massachusetts

Raymond Lathrop Barrett, Springfield.
1. George L. Steele; 2. Laurence D. Chapin; 3. J. H. Means and Roger I. Lee.

John A. Foley, Boston.
1. Soma Weiss; 2. William B. Castle; 3. J. H. Means and Roger I. Lee.

Allen Sheppard Johnson, Springfield.
1. Laurence D. Chapin; 2. George L. Steele; 3. J. H. Means and Roger I. Lee.

Egon Emil Kattwinkel, Auburndale.
1. Dwight O'Hara; 2. William D. Reid; 3. Roger I. Lee.

Jerome Andrew Whitney, Springfield.
1. George L. Steele; 2. Laurence D. Chapin; 3. James H. Means and Roger I. Lee.

Michigan

Russell L. Finch, Lansing.
1. Milton Shaw; 2. L. G. Christian; 3. James D. Bruce.

George Lawrence Leslie, Howell.
1. George A. Sherman; 2. Richard M. McKean; 3. James D. Bruce.

Harold Abraham Robinson, Detroit.
1. William G. Gordon; 2. Lawrence Reynolds; 3. James D. Bruce.

Harold Riche Roehm, Birmingham.
1. Walter M. Simpson; 2. Carl V. Weller; 3. James D. Bruce.

Ferdinand Ripley Schemm, Ann Arbor.
1. Cyrus C. Sturgis; 2. Carl V. Weller; 3. James D. Bruce.

Joseph Francis Whinery, Grand Rapids.
1. Joseph B. Whinery; 2. Thomas D. Gordon; 3. James D. Bruce.

Minnesota

Elmer Clarence Bartels, Duluth.
1. P. P. Vinson; 2. Fred W. Gaarde; 3. George E. Brown and E. L. Tuohy.

Vernon Lawrence Evans, Rochester.
1. James F. Weir; 2. George E. Brown; 3. Edward L. Tuohy.

Ross Martin Lymburner, Rochester.
1. F. A. Willius; 2. George E. Brown; 3. Edward L. Tuohy.

James Stuart McQuiston, Rochester.
1. George E. Brown; 2. Samuel F. Haines; 3. E. L. Tuohy.

Harold Conrad Ochsner, Rochester.
1. H. Milton Conner; 2. George E. Brown; 3. Edward L. Tuohy.

Edward George Thorp, Rochester.
1. F. W. Gaarde; 2. O. K. Maytum; 3. Edward L. Tuohy.

Mississippi

George Lamar Arrington, Meridian.
1. Noel C. Womack; 2. Felix J. Underwood; 3. G. W. F. Rembert.

Nevada

Lawrence Parsons, Reno.
1. John C. Ruddock; 2. William H. Leake; 3. Egerton Crispin.

New Jersey

Ferdinand Charles Dinge, East Orange.
1. John W. Gray; 2. George H. Lathrope; 3. W. Blair Stewart (deceased).

Paolo F. Liva, Lyndhurst.
1. Richard Edward Knapp; 2. Herman Trossbach; 3. W. Blair Stewart (deceased).

Carlyle Morris, Metuchen.
1. Frederick L. Brown; 2. F. C. Johnson; 3. W. Blair Stewart (deceased).

Joseph Reginald Pierson, Trenton.
1. Joseph T. Beardwood; 2. J. F. Pessel; 3. W. Blair Stewart (deceased).

New York

Victor W. Bergstrom, Binghamton.
1. H. B. Marvin; 2. John C. S. Lappeus (deceased); 3. James Alex. Miller and Robert A. Cooke.

Stephen H. Curtis, Troy.
1. Crawford R. Green; 2. Harry W. Carey; 3. Robert A. Cooke.

Maurice Coleman Harris, New York.
1. Samuel Weiss; 2. Robert Chobot; 3. Robert A. Cooke.

Warren F. Kahle, Larchmont.
1. Arthur F. Heyl; 2. Richard A. Kern; 3. O. H. Perry Pepper and Robert A. Cooke.

Max Mensch, Brooklyn.
1. Philip I. Nash; 2. Thomas J. Longo; 3. Robert A. Cooke.

Theresa Scanlan, New York.
1. C. F. Tenney; 2. Henry T. Chickering; 3. James Alex. Miller and Robert A. Cooke.

Isaac Shapiro, Schenectady.
1. Lester Betts; 2. Frank vander Bogert; 3. Robert A. Cooke.

George Widmer Thorn, Buffalo.
1. Nelson G. Russell; 2. Clayton W. Greene; 3. Allen A. Jones.

Max Trubek, New York.
1. Louis F. Bishop, Jr.; 2. Louis F. Bishop, Sr.; 3. James Alex. Miller and Robert A. Cooke.

North Carolina

George Curtis Crump, Asheville.
1. A. B. Craddock; 2. S. L. Crow; 3. C. H. Cocke.

Paul Allison Yoder, Winston-Salem.
1. Wingate M. Johnson; 2. P. P. McCain; 3. C. H. Cocke.

Ohio

Abel A. Applebaum, Toledo.
1. L. A. Levison; 2. John T. Murphy; 3. A. B. Brower.

Frank J. Doran, Cleveland.
1. Ralph K. Updegraff; 2. Henry J. John; 3. A. B. Brower.

Oregon

George Wilber Millett, Portland.
1. John H. Fitzgibbon; 2. Marr Bisaillon; 3. Homer Coffen.

Pennsylvania

Nathan Blumberg, Philadelphia.
1. Joseph C. Doane; 2. Arthur C. Morgan; 3. E. J. G. Beardsley.

John Milnes Dyson, Hazleton.
1. David Riesman; 2. Charles H. Miner; 3. O. H. Perry Pepper and E. J. G. Beardsley.

Paul H. Parker, Jenkintown.
1. Joseph T. Beardwood; 2. H. L. Bockus; 3. William D. Stroud and E. J. G. Beardsley.

Floyd W. Stevens, Scranton.
1. Arthur E. Davis; 2. W. M. Donavan; 3. George Morris Piersol.

Elwood Wakefield Stitzel, Altoona.
1. E. Roland Snader, Jr.; 2. Augustus S. Kech; 3. E. Bosworth McCready.

Rhode Island

Henry Stephen Joyce, Providence.
1. Charles F. Gormly; 2. Guy W. Wells; 3. Alex. M. Burgess.

Clifton Briggs Leech, Providence.
1. Charles F. Gormly; 2. Guy W. Wells; 3. Alex. M. Burgess.

Harvey Elijah Wellman, Providence.
1. Charles F. Gormly; 2. Guy W. Wells; 3. Alex. M. Burgess.

Henry L. C. Weyler, Providence.
1. Charles F. Gormly; 2. Guy W. Wells; 3. Alex. M. Burgess.

Elihu Smith Wing, Providence.
1. John F. Kenney; 2. Charles F. Gormly; 3. Alex. M. Burgess.

Tennessee

Tolbert Clinton Crowell, Chattanooga.
1. Franklin B. Bogart; 2. Leopold Shumacker; 3. J. O. Manier.

Texas

Edgar Mullins Dunstan, Dallas.
1. Hugh Leslie Moore; 2. C. M. Grigsby; 3. C. T. Stone.

William J. Marr, Jr., Galveston.
1. George Herrmann; 2. George E. Bethel; 3. C. T. Stone.

Wendell Heath Paige, Brownwood.
1. Will S. Horn; 2. T. C. Terrell; 3. C. T. Stone.

Virginia

Vincent Joseph Dardinski, Clarendon.
1. Eugene R. Whitmore; 2. Walter Freeman; 3. Wallace M. Yater and J. Morrison Hutcheson.

Canal Zone

Ernst Thonnard-Neumann, Cristobal.
1. C. D. Briscoe; 2. Tomas Guardia; 3. William M. James.

Hawaii

Arthur Warren Duryea, Honolulu.
1. Harry L. Arnold; 2. A. G. Schnack.

Medical Corps, U. S. Army

Major Coleridge Livingstone Beaven, Washington, D. C.
3. Robert U. Patterson.

Major Samuel McPherson Browne, Washington, D. C.
1. Leon A. Fox; 2. Roger Brooke; 3. Robert U. Patterson.

Lt. Col. Frederick Hultman Foucar, San Francisco, Calif.
3. Robert U. Patterson.

Major Arthur Raymond Gaines, Denver, Colo.
1. William C. Pollock; 2. Everett L. Cook; 3. Robert U. Patterson.

Captain Horace Page Marvin, Denver.
1. William C. Pollock; 2. Everett L. Cook; 3. Robert U. Patterson.

Major Joseph Aaron Mendelson, Cheyenne, Wyoming.
3. Robert U. Patterson.

Medical Corps, U. S. Navy

Lt. Comdr. Lyle Jay Roberts, Washington, D. C.
1. Walter M. Anderson; 2. E. R. Stitt; 3. P. S. Rossiter.

U. S. Public Health Service

Olis Leon Anderson, Chelsea, Mass.
1. Albert D. Foster; 2. Frederick C. Smith; 3. J. P. Leake and Hugh S. Cumming.

Canada

Alberta

Percy Harry Sprague, Edmonton.
1. F. A. Willius; 2. Albert M. Snell; 3. Edward L. Tuohy.

Korea

Chosen

Zacharias Bercovitz, Pyengyang.
1. John Mark Lacey; 2. John V. Barrow; 3. Egerton Crispin.

On motion seconded and regularly carried, it was

RESOLVED, that the above 93 candidates shall be and are herewith elected to Associateship in the American College of Physicians.

REPORT OF THE TREASURER

The Treasurer, Dr. William D. Stroud, presented the financial report in the absence of Dr. Charles F. Martin, Chairman of the Finance Committee, who was unable

to attend because of illness. Dr. Stroud reported that five dividends had been received during the current year from closed banks in Pittsburgh, which reduces our balance in those institutions to $11,900.00, whereas the original amount had been approximately $37,000.00. The prospects are that there will be little or no loss from those banks. Dr. Stroud then reported that $4,000.00, par value, Bonds of the City of Detroit had defaulted this year in their interest payments, but that the bonds had been deposited in accordance with the recommendations of our bank on a refinancing plan, which will probably result in the issuance of new bonds and the payment of past interest on the old bonds. The Treasurer also reported that since the last meeting of the Board of Regents, $5,000.00 Province of Ontario bonds had been called, and that the proceeds were available for reinvestment. On motion seconded and regularly carried, it was

RESOLVED, that the Treasurer be authorized, after consultation with the Finance Committee, to reinvest proceeds of the $5,000.00 maturity, Province of Ontario bonds, plus any other amounts not deemed necessary for the current expenses of the College.

In discussion that followed, it was suggested that possibly Government Bonds or Treasury Certificates would be appropriate securities in which to invest any surplus funds of the College at the present time.

The Treasurer then mentioned the fact that while the dues have been reduced practically 25 per cent for 1933 and our income thereby reduced approximately $9,000.00, the number of delinquencies in dues has not been appreciably affected thereby, and that as many members are actually delinquent as when the dues were higher.

The Treasurer then presented two proposed budgets for 1934, one for the office of the General Chairman of the Annual Clinical Session and the other for the office of the President, in connection with his program for the Annual Clinical Session. These budgets covered expenses of secretarial service, honoraria to the banquet speaker, expenses for the publicity committee, the Ladies Entertainment Committee, traveling expenses for invited guest speakers, etc. These budgets were prepared in accordance with the directions of the Board of Regents at their meeting a year ago.

The Treasurer then presented financial reports prepared by the Executive Secretary. One report consisted of a comparison of the 1932 expenditures, the 1933 budget and the 1933 expenditures, with the months of November and December estimated. This statement disclosed that the total expenditures in 1932 amounted to $61,376.58, that the budget for 1933 was reduced to $50,686.67, and that the actual expenditures for 1933 will be approximately $47,255.35. This is a reduction of $14,121.23 from the expenditures of 1932 and is less than the budget appropriation for 1933 by $3,431.32. The other financial statement presented to the Board of Regents showed the income and expenses from January 1, 1933, to October 31, 1933, and the estimated income and expenses from November 1, 1933, to December 31, 1933. The anticipated surplus for 1933, shown on this statement, was $5,765.23. The Treasurer then asked the Executive Secretary, Mr. Loveland, to analyze the statements presented, which he did. Mr. Loveland pointed out to the Board of Regents that the income for 1933 had been greatly reduced for the following reasons:

(1) Dues had been reduced approximately 25 per cent;

(2) No elections to Fellowship or Associateship have taken place since the Annual Clinical Session at Montreal, whereas customarily there is a meeting of the Board of Regents in the middle of the year, when new elections take place and the fees and dues from such new members go into the year's income. This year, for

the sake of economy, the Board of Regents was not called together as many times as usual, and with the present meeting at the end of the year, most of the income arising from the new elections will go into next year's business;

(3) The change in the By-Laws requiring new candidates to be presented first for Associateship, has very greatly reduced the number of possible candidates for Fellowship. Whereas the 1931 income from Fellowship initiation fees amounted to $18,365.00, the income from that source for the present year to date has amounted only to $2,040.00;

(4) Financial stringencies of the times are responsible for a greater degree of delinquency in dues and in failure of subscribers to the Life Membership Fund to pay their annual subscriptions. For illustration, in 1931 income from subscriptions to the Life Membership Fund amounted to $2,400.00, in 1930 to $3,100.00, whereas the amount received to date this year was $425.00;

(5) Income from bonds and income from interest on bank balances have been reduced due to the fact that we received no income on the balances in closed banks, that we received no income on balances in checking accounts since March 1933, and that $4,000.00 of the Bonds of the City of Detroit defaulted in interest payments.

Strict economy has been practiced by all Officers of the College this year, and the prospect of finishing the year with a surplus appears to be gratifying indeed under the conditions outlined above.

By resolution, the report of the Treasurer and the Executive Secretary was approved.

Report of the Committee on the John Phillips Memorial Prize

The Chairman of the Committee on the John Phillips Memorial Prize, Dr. David P. Barr, reported that the method of procedure in the selection of candidates for the 1934 award had been followed as previously, but that after a very careful survey by his Committee, there appeared to be no outstanding favorite among the candidates submitted, and that no exceedingly outstanding or meritorious piece of work had been completed during the past year by any of those under consideration. The Committee also felt that the Board of Regents might appropriately more definitely determine what the prize shall be in the future. The Committee presented the following recommendations:

(1) That the Phillips Memorial Prize be not awarded this year;

(2) While the directions to the Committee are specific as to the mode of conducting the competition for the prize, the Committee has no instructions as to what the prize shall be. It is recommended that the Board of Regents at their next meeting definitely establish what the policy shall be with regard to the exact nature and form of the award.

Thereafter the following resolutions were adopted:

Resolved, that the above recommendation (1) of the Committee on the John Phillips Memorial Prize be approved;

Resolved, that the present Committee on the John Phillips Memorial Prize formulate and present at the next meeting of the Board of Regents a recommendation with regard to the exact nature and form of the Phillips Memorial award.

Report of the Editor of the Annals of Internal Medicine

The Editor of the Annals of Internal Medicine, Dr. Maurice C. Pincoffs, reported that in accordance with authority granted by the Board of Regents he and the Executive Secretary, after making an extensive study of various printers, had selected the Lancaster Press, of Lancaster, Pa., beginning with the July 1933 number. This concern prints almost exclusively scientific journals and books. A considerable saving has been effected, and improved service has been obtained. The format of the journal has been changed, with the apparent approval of every one. A large number of manuscripts are received, there being an ample supply for some months to come. Approximately one in four of the manuscripts submitted is accepted. The work of the Editor's Office is carried on with the printers from two to three months in advance.

Discussion followed concerning the continuation of the publication of the present Editorial Council on the back cover of the Annals. This Council has not been an active one in recent years, and has probably been carried over from the time the late Dr. Warthin was Editor. General discussion disclosed that the present Committee on the Annals of Internal Medicine, consisting of three members, is a standing Committee having general responsibility for the Annals in questions relating to administration and publication. The thought was expressed that the present Committee might be modified to provide for five members, and that these five members need not necessarily be members of the Board of Regents. The present Editorial Council should be abolished and an active one appointed to function not only in relation to administration and publication, but also in relation to the publication of editorials, the selection of articles published and to share with the Editor the responsibility of rejecting manuscripts.

President Piersol summed up the discussion and expressed the consensus of opinion as being that the Editor of the Annals should be asked to get in touch with his Committee and at the next meeting of the Board of Regents to be ready to give recommendations for the modification of the present Committee, or some other plan for the appointment of an Editorial Council.

Report of the Committee on Public Relations

The Chairman of the Committee on Public Relations, Dr. Charles G. Jennings, presented reports on two cases involving contract practice and advertising. The Board of Regents directed the Committee to make a further and more complete study of the cases and report at the next meeting of the Board of Regents.

Old and New Business

President Piersol reported that on September 14, 1933, in accordance with provisions of the By-Laws, he had appointed the following members of the Board of Governors to serve until the next regular election:

Dr. Robert B. Kerr (Fellow), Manchester, Governor for New Hampshire;
Dr. Clarence L. Andrews (Fellow), Atlantic City, Governor for New Jersey.

These appointments were occasioned by the death of Dr. Edward O. Otis, Governor for New Hampshire, and by the death of Dr. W. Blair Stewart, Governor for New Jersey. Dr. Ernest B. Bradley, Vice Chairman of the Board of Governors, now succeeds to the Chairmanship of the Board of Governors and becomes ex officio a member of the Board of Regents, until the next regular election.

On behalf of the Nominating Committee, the Executive Secretary asked the wishes of the Board of Regents regarding the nomination of a Governor of the Col-

lege for Puerto Rico. The College has a few members in Puerto Rico, and there has been a recommendation that an official Governor should be elected to facilitate the presentation of candidates from that Island. The Board concurred in the opinion that the Nominating Committee should nominate a Governor for Puerto Rico.

The Executive Secretary reported that the resignations of Dr. James J. Gable (Associate), Norman, Okla., and Dr. Everett E. Watson (Associate), Salem, Va., held over from the last meeting of the Board of Regents, had been withdrawn and their active memberships reëstablished.

The Executive Secretary then reported that Dr. Ray G. Barrick (Associate), Joliet, Ill., and Dr. James E. Harvey (Associate), Pasadena, Calif., had been dropped from the roll because of failure to take up their election in the prescribed period of one year.

The Executive Secretary was authorized to dispense with the services of an official medical reporter for the general scientific sessions at the next Clinical Session.

The following resolution was adopted:

RESOLVED, that the matter of the adoption of a Life Membership Certificate be referred to a Committee of three, who shall report to the Board of Regents at their next meeting.

Dr. Walter L. Bierring reported that the Council on Medical Education and Hospitals of the American Medical Association had been requested to investigate the entire subject of specialization and make recommendations, looking to the establishment of proper qualifications of physicians who shall engage in special practice and that the report of the Council and its recommendations be submitted to the House of Delegates as soon as practicable. In compliance with these instructions, after a two year study, the Council had determined that it seemed desirable to bring together, for mutual discussion, some of those who are interested in various phases of this problem, and a preliminary meeting had been held at Milwaukee on Sunday, June 11, at the Hotel Wisconsin. The conference was arranged jointly by some of the special examining boards, by the National Examining Board and by the Council. Dr. William D. Cutter, of the Council on Medical Education and Hospitals of the American Medical Association, invited the American College of Physicians to send a representative to that meeting, and President Piersol had asked Dr. Bierring to represent us, although Dr. Bierring was officially there on behalf of the Federation of State Licensing Boards. Among other organizations represented were the American Association of Medical Colleges, the Council on Medical Education and Hospitals of the American Medical Association, the National Examining Board, the American Board of Ophthalmology, the American Board of Otolaryngology, the American Board of Obstetrics and Gynecology, the Board for Pediatrics, the Board for Orthopedics, the Board for Dermatology, the Board for Proctology and the Board for Psychiatry. Dr. Louis Wilson, of Rochester, Minn., presided. Dr. Paul Titus, of Pittsburgh, acted as Secretary, and Dr. W. P. Wherry, of Omaha, was in charge of arrangements. The meeting resulted in general discussion of a means for the certification and licensure of specialists in the various fields of medicine. Suggestions for a constitution of an "Advisory Council on Medical Specialists" were presented and further meetings scheduled for future development of this Advisory Council.

In the general discussion that followed, the consensus of opinion was that the American College of Physicians should take immediate action looking toward its participation in the certification of internists and others engaged in affiliated specialties. The College is the natural body to function in this field, and the Board of Regents might initiate written examinations, for which machinery is already set up

in the By-Laws for the admission of candidates to Fellowship, and might further initiate additional examinations for certification of physicians as specialists in internal medicine and affiliated specialties. On motion seconded and regularly carried, it was

RESOLVED, that the Chair appoint a Committee of three to make a complete and thorough study of this situation and report back at the next meeting of the Board of Regents.

The Secretary-General, Dr. William Gerry Morgan, read a communication from Surgeon General Robert U. Patterson, of the U. S. Army, concerning the support by various medical organizations of the project for the building of a new library for the Medical Department of the Army, in connection with the Walter Reed Medical Center. The recommendation of the Board of Regents was that this is a worthy project and should be encouraged, and suggested that the Secretary-General write to the proper officials advocating the building of the library but refraining from suggesting the source from which funds therefor should be taken, because this is not in the domain of the College.

Secretary-General Morgan then read a communication from Dr. William H. Walsh (Fellow), Chicago, presenting a proposed plan for a Council on Professional Service and Administrative Practice. The proposal provided that the Council should be specifically directed to the problems involving various economic phases of practice, methods and procedures for the certification of specialists, standards for the practical training of technicians, dietitians, clinical record librarians in hospitals, etc.

On behalf of the Chairman of the Committee on Clinics, Dr. Arthur R. Elliott, the Executive Secretary presented for the consideration of the Board the matter of an official scientific exhibit at future annual meetings. Dr. Elliott had pointed out that it is probably desirable to forego such an exhibit this year but recommended that the idea of a systematic scientific exhibit in the future should be considered by the Board of Regents as one of the principal activities of the College.

President Piersol at this point presented an invitation from Philadelphia to the American College of Physicians to meet in that City during 1935.

Adjournment.

Attest: E. R. LOVELAND,
Executive Secretary.